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DEVELOPMENT OF THE OTIC CAPSULE

III. FETAL AND INFANTILE CHANGES IN THE FISSULAR REGION AND THEIR PROBABLE RELATIONSHIP TO THE FORMATION OF OTOSCLEROTIC FOCI

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The fissula ante fenestram, a channel of connective tissue extending from the vestibule at its junction with the scala vestibuli through the otic capsule in front of the oval window to the middle ear, is a constant structure in the human ear. It was first referred to by Huschke [1] in 1844, by Hyrtl [2] in 1845 and by Siebenmann [3] in 1890. The early literature on this structure was summarized by me [4] in 1930 and need not be cited here. Since 1930 the fissula has been studied more extensively. In 1933 I [5] described its development from the earliest embryologic appearance to full term. Anson and Wilson [6] in 1933 described the fissula in an adult human ear, and Anson and Martin [7] in 1935 and Wilson [8] in 1935 discussed its form, contents, variations

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1. Huschke, E., in Sömmerring: Lehre von den Eingeweiden und Sinnesorganen, Leipzig, Leopold Voss, 1844, vol. 5, p. 828.

2. Hyrtl, Joseph: Vergleichend-anatomische Untersuchungen über das innere Gehörorgan des Menschen und der Säugethiere, Prague, F. Ehrlich, 1845.

3. Siebenmann, Fredrich: Die Korrosions-Anatomie des knöchernen Labyrinthes des menschlichen Ohres, Wiesbaden, J. F. Bergmann, 1890.

4. Bast, T. H.: Ossification of the Otic Capsule in Human Fetuses, Contrib. Embryol. (no. 121) **21**:53 (June) 1930.

5. Bast, T. H.: Development of the Otic Capsule: II. The Origin, Development and Significance of the Fissula Ante Fenestram and Its Relation to Otosclerotic Foci, Arch. Otolaryng. **18**:1 (July) 1933.

6. Anson, B. J., and Wilson, J. G.: The Fissula Ante Fenestram in an Adult Human Ear, Anat. Rec. **56**:383 (July 25) 1933.

7. Anson, B. J., and Martin, J.: Fissula Ante Fenestram: Its Form and Contents in Early Life, Arch. Otolaryng. **21**:303 (March) 1935.

8. Wilson, J. G.: Fissula Ante Fenestram and the Adjacent Tissue in the Human Otic Capsule, Acta oto-laryng. **22**:382, 1935.

and deformations in early life. Considerable variations in the size and shape of the fissula ante fenestram do occur, and they have been described by Anson and Martin,[7] Wilson [8] and myself.[5] The present observations agree entirely with the descriptions just mentioned, and size and shape will not be discussed in detail here.

The purpose of this paper is to describe a peculiar type of cartilaginous or osteoid mass which develops in the otic capsule and which may lie next to the fissula, surround it or partially obliterate it. Since the fissula region of the otic capsule is a site of predilection for the otosclerotic foci, an understanding of the early histologic changes and variations of this area seems of importance.

The observations reported here were made on serial sections, and models thereof, of thirty-two ears from human fetuses ranging in age from 21 weeks to full term and of twenty ears from children ranging in age from 3 days to 6 years.

Anson and Martin [7] and Wilson [8] described a number of children's ears in which a cartilage mass or, in the ears of some older persons, an osteoid mass partly or wholly plugged the fissula ante fenestram or lay adjacent to it. My observations are similar to theirs but were made to a large extent on ears of fetuses and young children. This account, therefore, is intended as an embryologic approach to the problem so well presented by Anson and Martin [7] and Wilson.[8]

The cartilage masses in question make their appearance in older fetuses. The earliest stage at which one was noted was in a fetus of 180 mm. crown-rump length, or at a fetal age of about 21 weeks. At this age the whole otic capsule is ossified except for the region of the lateral canal and the narrow rims around both the stapes and the fissula ante fenestram, which are still cartilage.[4] The rim around the fissula, however, at least in the majority of cases, seems quiescent or dormant and can be seen even in adult ears. The specific cartilage mass, therefore, which is seen in the fissular region in only a certain percentage of ears from older fetuses and infants can hardly be considered normal or at least is not a usual stage in the development of the capsule. This raises a number of questions:

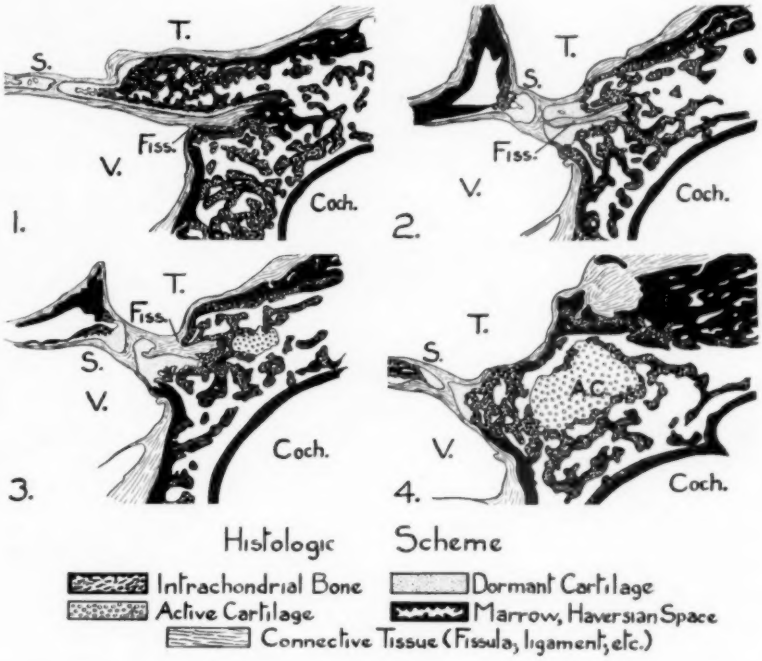
1. Is there a normal histologic picture of the fissular region of the otic capsule?

2. What are the structure, origin and fate of the cartilage mass? Is it derived from the fissular tissue or from the rim of cartilage around the fissula, and as it grows does it invade the surrounding bone or does it reduce the size, alter the shape or destroy the integrity of the fissula ante fenestram?

3. In what percentage of ears of infants and fetuses do these changes occur?

4. Is there any relation between the process and the formation of otosclerotic bone in later life?

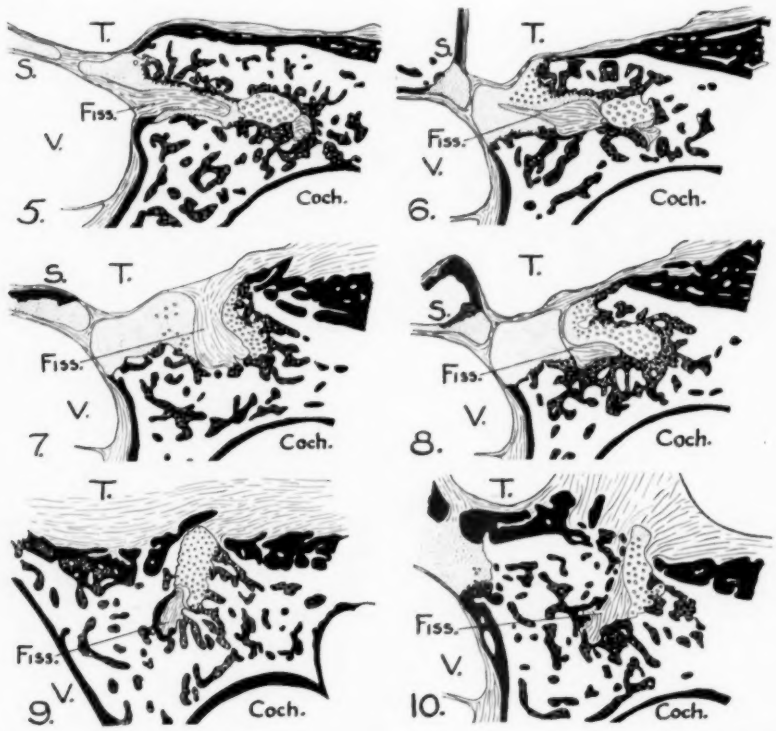
The first of these questions, as to whether there is a normal histologic picture of the fissular region of the otic capsule, was in part answered in a previous paper.[5] In short it was stated in that paper that by the twenty-second or twenty-third week of fetal life the entire otic capsule is ossified except for an area in the region of the lateral



Figs. 1 to 4 (embryo 45A, aged 29 weeks, 260 mm. crown-rump length).—Drawings of horizontal sections at four levels through the fissular region of the capsule. A model of the fissula is shown in figure 49. The fissula is open throughout, with the tympanic opening close to the stapedial joint (fig. 3). A dark-stained mass of active cartilage fills the aberrant upper extension of the fissula *A.C.* (figs. 3 and 4). Intrachondrial bone is forming at the margin of the mass. There is much dormant cartilage at the stapedial joint. In this and the following figures *T.* indicates the tympanum; *V.*, the vestibule; *S.*, the stapes; *Fiss.*, the fissula ante fenestram; *Coch.*, the cochlea, and *A.C.*, the mass of active cartilage.

canal and a rim around the oval window. There also is a ring of cartilage around most of the fissula, but this remains essentially unchanged throughout life. Thus, the fissula, which consists of connective tissue, is more or less surrounded by a thin rim of cartilage, and this in

turn is surrounded by the bone of the otic capsule. This is the general histologic picture of the fissular region as seen in fetuses and children; however, one hesitates to speak of a given histologic picture as normal, since so many variations occur that no two look alike. For example, the fissula may be large and massive, or it may be small and slitlike. In some specimens the cartilage surrounding the fissula may be thin or even absent in places, while in others the cartilage is thick and completely surrounds the fissula. The surrounding bone, too, varies greatly.



Figs. 5 to 10 (embryo 70, aged 23 weeks, 202 mm. crown-rump length).—Drawings of horizontal sections at six levels through the fissular region of the capsule. A model of this fissula is shown in figure 47. The fissula is open throughout. A dark-stained mass of active cartilage is present along the entire cochlear margin of the fissula and is shown in all six sections.

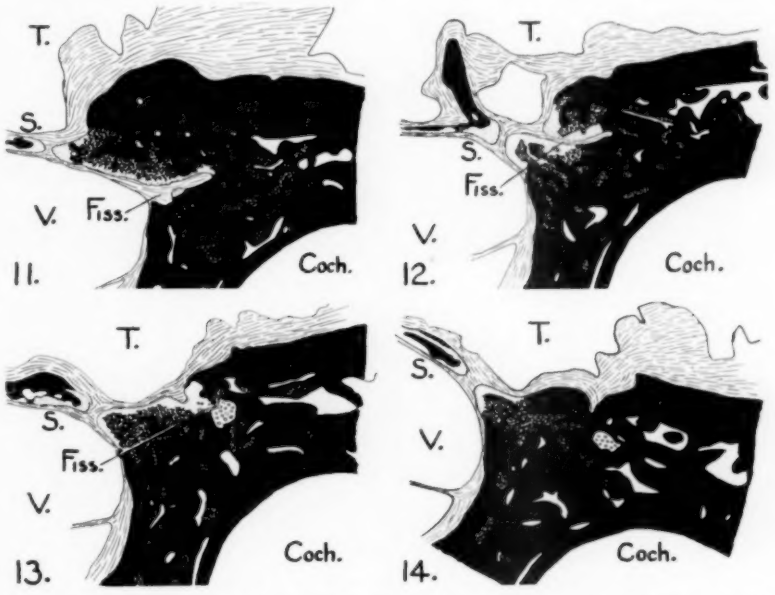
In some cases the bone is dense, with few marrow spaces, while in others the marrow is abundant and the bone trabeculae are small. In some specimens much intrachondrial bone (so-called cartilage islands) is present, while in others most of the formation is endochondral bone. Thus, the term normal fissular region can at best express only a general idea. Examples of what is here regarded as a normal fissular region at various ages are shown in figures 23 to 30.

The narrow rim of cartilage between the fissular tissue and the bone is not apparent because of the low magnification of the sections.

STRUCTURE, ORIGIN AND FATE OF THE CARTILAGE MASS

In considering the structure, origin and fate of the cartilage mass the following questions arise: Is it derived from the fissular tissue or from the rim of cartilage around the fissula, and as it grows does it invade the surrounding bone or does it reduce the size, alter the shape or destroy the integrity of the fissula?

As already mentioned, the cartilage which lies between the fissula and the bone of the capsule is cellular and young-looking, yet has every



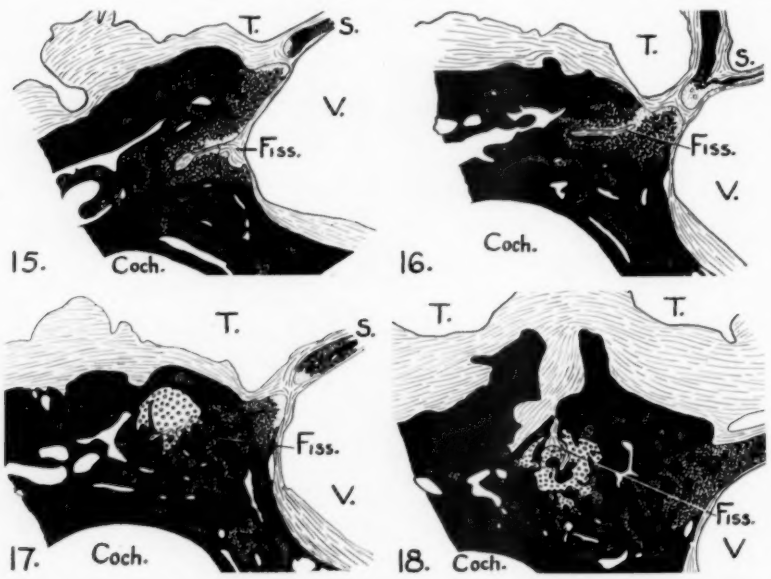
Figs. 11 to 14 (infant 93L, aged 14 months).—Drawings of horizontal sections at four levels through the fissular region of the capsule. The fissula is open throughout, but the cartilage mass is present in the aberrant upward extensions of the fissula (figs. 13 and 14). There is considerable intrachondrial bone between the fissula and stapedial joint.

appearance of resting, or dormant, cartilage; and it is much the same in older fetuses as in the adult. Anson and Martin referred to it as adult cartilage. However, because of its cellular character and its lack of nests of cells, because it may occur in the fetus as well as in the adult and because the cartilage may undergo further change, I suggest the term dormant cartilage instead of adult cartilage.

The cartilage mass which is the topic of the discussion stands in contrast to the dormant fissular cartilage in that the cells are large

and the matrix is more prominent, staining lightly with hematoxylin in some sections and deeply in others. This cartilage apparently is in the late stages of development, just prior to the invasion of osteogenetic tissue and replacement by bone.

Such a mass of active cartilage apparently arises in and from the dormant fissular cartilage, which may be augmented by new cartilage derived from the fissular tissue by metamorphosis of its connective cells to cartilage cells. The cartilage mass is formed by enlargement of the cells of the fissular cartilage; then the matrix changes character and is deeply stained by hematoxylin. This is exactly what happens in any other cartilage just prior to the invasion by osteogenetic buds.

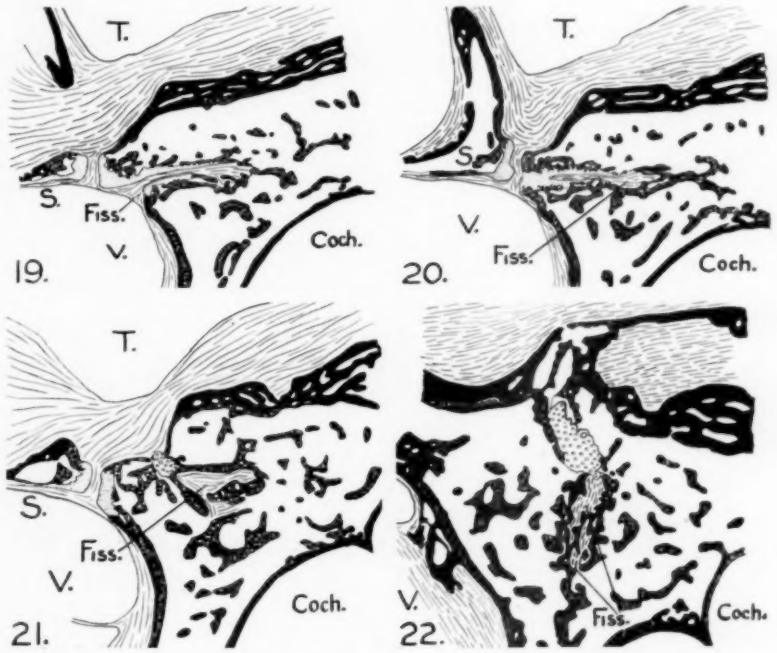


Figs. 15 to 18 (infant 93R, aged 14 months).—Drawings of horizontal sections at four levels through the fissular region of the capsule. A cartilage mass which is partly ossified fills the upper aberrant extension of the fissula and has encroached on the fissula, making it small and very irregular. There is much intrachondrial bone around the fissula. A model of the fissula is shown in figure 50.

It appears, then, that the cartilage is older than the fissular cartilage and has reached the final stage preceding the process of ossification. That this is the case is indicated in some of the older ears, in which the cartilage mass is being replaced by osteoid tissue or even by intrachondrial bone. Anson and Martin observed and recorded this process. The question raised here is therefore one only of terminology and not one of concept.

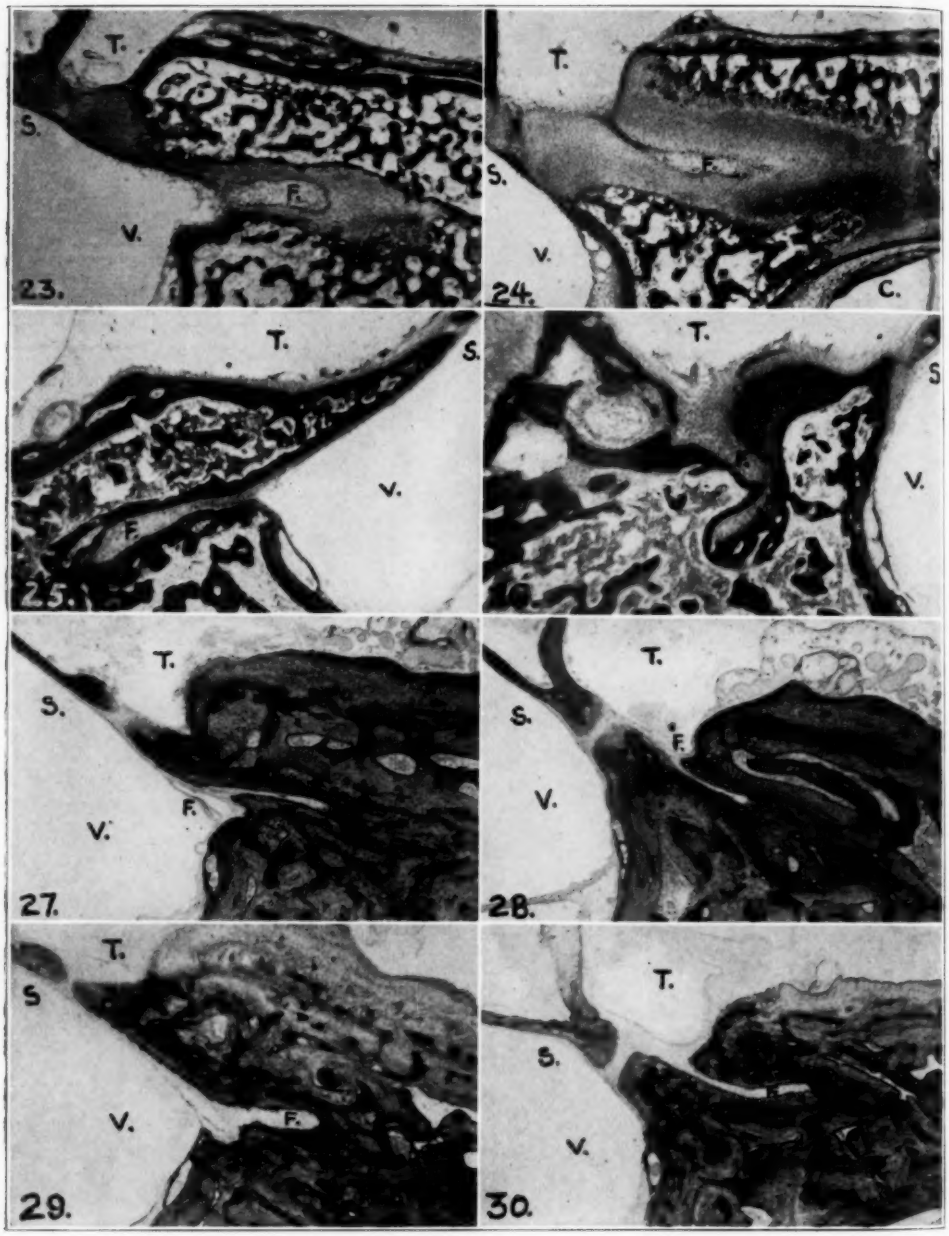
The question as to whether the mass of active cartilage invades the surrounding bone is here answered in the negative. In the series of ears studied I have seen no evidence of erosion and invasion of bone by

this cartilage. Of course, as the cartilage cells enlarge, room is needed, and the mass will bulge into any soft tissue area adjacent to it. Thus it may bulge into bone marrow spaces; it will narrow the fissula or even obliterate it, and if it is in the region of the tympanic opening of the fissula it will bulge into the tympanic cavity (fig. 39). The bulging is a necessary result of the enlargement of the cartilage cells, but I find no evidence in my preparations that the expanding process involves the surrounding bone until after the ossification of the cartilage mass has begun, when reexcavation and rebuilding of the surrounding bone may occur, as is noted in older ears (figs. 35, 37 and 38).



Figs. 19 to 22 (embryo 62, aged 24 plus weeks, 2.5 mm. crown-rump length).—Drawings of horizontal sections at four levels through the fissula. A plug of cartilage fills the tympanic opening of the fissula, bulges into the tympanic cavity and extends upward along the tympanic margin of the aberrant upward extension of the fissula, but without abolishing it (figs. 21 and 22). The fissula and cartilage mass are shown in the model in figure 48.

Location of the Mass of Active Cartilage.—It is well known that the fissula differs both in size and in shape in the fetal ears. In some specimens the fissula is small and narrow, while in others it may be very broad, extending from the vestibule almost to the cochlea (fig. 31). In other specimens the fissula is very tall, extending far above its opening to the middle ear. It is probable that in the development the process of formation of fissular tissue goes beyond bounds and forms a



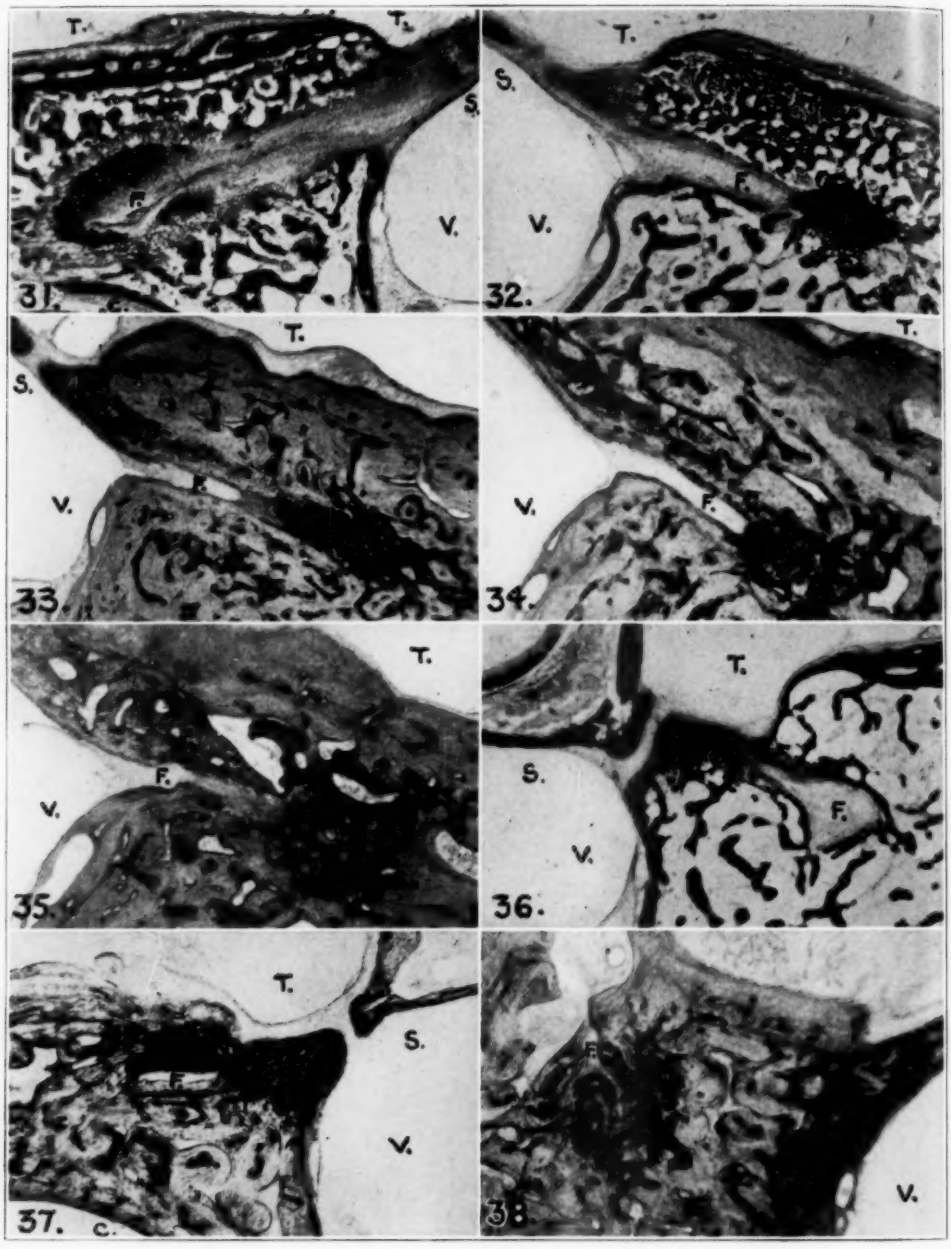
Figs. 23 to 30.—Photographs of sections through normal fissular regions of ears of subjects of different ages. The sections shown in figures 23, 25, 27 and 29 are at the level of the vestibular opening, and those in figures 24, 26, 28 and 30 are at the level of the tympanic opening of the fissula. Figures 23 and 24 present sections from the left ear of embryo 21 (aged 21 weeks); figures 25 and 26, from the right ear of embryo 2 (aged 26 weeks); figures 27 and 28, from the left ear of infant 115 (full term), and figures 29 and 30, from the left ear of infant 93 (aged 14 months).

fissula larger than necessary and that later an attempt is made to reduce the size, a process which may also be carried to excess, obliterating parts of the fissula.

This interpretation permits a correlation of large fissulae with the formation of cartilage masses. From a study of my series of ears it has become appparent that in most instances the mass occurs in the part of the fissula which is unusually large. The earliest instances are observed in fetuses of about 21 weeks and older. In these early stages the mass is found (1) along the cochlear margin of the fissula, (2) in the aberrant fissular extension above the tympanic opening of the fissula or (3) in the fissula at its tympanic opening. In older fetuses it may also occur (4) in the region where the fissula extends toward the stapedial joint. In the first three situations the cartilage mass is limited, circumscribed and tumor-like, while in the fourth situation the process appears to be more like a delayed process of formation of intrachondrial bone.

Changes Along the Cochlear Margin of the Fissula: These changes apparently occur in unusually deep fissulae. An example of this type is shown in figure 31, which depicts a specimen taken from a 180 mm. fetus, in which the fissula extends from the vestibule almost to the cochlea. At the cochlear end the cartilage surrounding it has already partly changed to the large cell type and is deeply stained with hematoxylin and eosin. In figure 32 (a section from the ear of embryo, 70, 202 mm. crown-rump length) the formation of the cartilage mass is well advanced, while in figures 33 and 34 the mass is undergoing ossification around the margin. In figure 35 (a section from a 10 week old infant) the mass is ossified. In these cases, then, the fissula was not destroyed by the process but only narrowed. Figures 5 to 10 are a series of sketches of sections of the ear of embryo 70, made at different levels through the fissular region to show the extent of the cartilage mass along the entire cochlear portion of the fissula. The remaining portion of the fissula is well formed and open throughout. The only effect of the cartilage mass on the fissula is to narrow it. Figure 47 is a model of this fissula and the adjacent cartilage mass.

Changes in the Aberrant Fissula Above Its Tympanic Opening.—Formation of a mass of active cartilage in the aberrant extension of the fissula above its tympanic opening apparently also develops in an effort to reduce the abnormally large part of the fissula. An example of this condition is shown in figures 1 to 4 and 11 to 18 and in the models shown in figures 49 and 50. In a number of cases the change occurs only in this aberrant upward extension, and the rest of the fissula seems normal. In some cases, however, the obliterating process extends downward and involves part or all of the tympanic opening of the fissula. This condition will be discussed under the next heading.

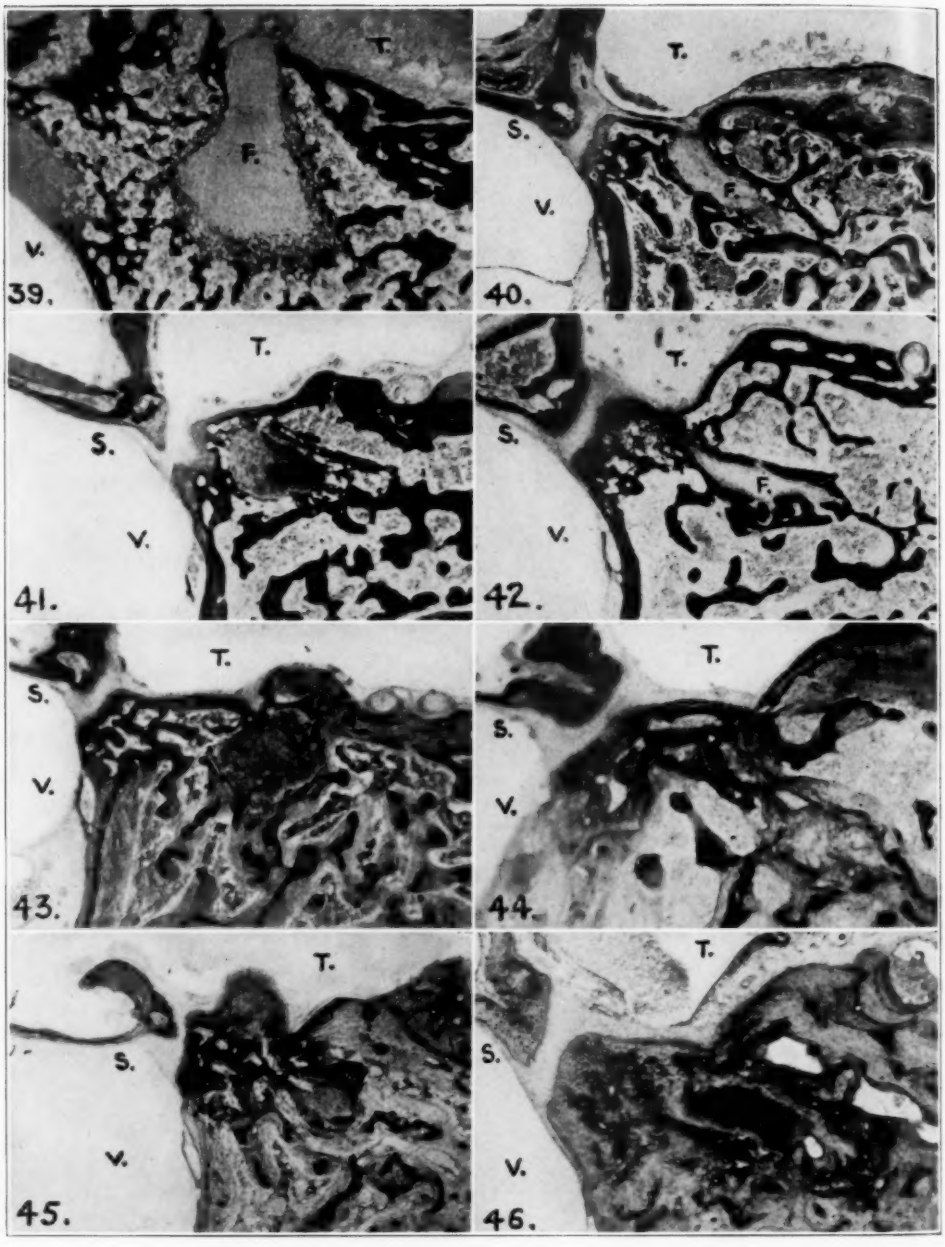


Figs. 31 to 38.—Photographs of sections through the fissular regions of ears of subjects of various ages. Figures 31 to 35 show a cartilage or osseous mass at the cochlear margin of the fissula. Figures 36 and 35 show localized ossification between the fissula and the stapedial joint. Figure 38 shows a localized osseous mass around the upper part of the fissula, where it opens into the middle ear. Figure 31 shows a section from embryo 29B (aged 21+ weeks), right ear; figure 32, from embryo 70 (aged 23 weeks), left ear; figure 33, from infant 103 (aged 1 year, 10⅔ months), left ear; figure 34, from infant 96L (aged 3 years), left ear; figure 35, from infant 83 (aged 10 weeks), left ear; figure 36, from embryo 46 (aged 25 weeks), left ear; figure 37, from infant 93R (aged 14 months), right ear, and figure 38, from infant 93R (aged 14 months), right ear.

Changes in the Fissula at Its Tympanic Opening.—In a considerable number of specimens there is a partial or complete blocking of the fissula at or near its tympanic opening by the mass of active cartilage or, in older specimens, by an osseous mass. The origin of such an occluding process may occur (1) by the downward growth of the cartilage mass in the aberrant part described in the previous paragraph or (2) by the formation of a mass of active cartilage in the tympanic opening of the fissula. Figures 39 to 46 are photomicrographs of sections of ears from subjects of different ages in which the tympanic opening is blocked by such a cartilage or bony mass. The first type is shown by the model in figure 50 and by the drawings of sections at various levels through the fissular region (figs. 15 to 18). It will be noted that the aberrant fissula is largely filled with cartilage partly changed to intrachondrial bone and that the process has extended downward and blocked most of the tympanic opening, leaving only three small points of communication with the tympanum.

The second type seems to be more common. The cartilage mass arises in the fissula at the tympanic opening. Again it is possible that the active cartilage develops in an attempt to reduce the large tympanic opening. In young fetuses a large slitlike tympanic opening of the fissula is sometimes seen, extending from the stapedial joint to, and beyond, the semicanal for the tensor tympani muscle. In older fetuses the ossifying process has usually limited the fissular opening so that it may occur as a small slit near the stapedial joint, somewhere between the stapedial joint and the semicanal for the tensor tympani muscle or into the semicanal. In some instances, however, the large tympanic opening is not reduced by the normal ossifying process, and therefore a delayed attempt to reduce the opening is made. This apparently is the purpose of the mass of active cartilage. Frequently, such a mass develops beyond bounds and closes the entire tympanic opening of the fissula. Figure 48 shows a model of such a fissula and a cartilage mass plugging the tympanic opening from the stapes to the semicanal of the tensor tympani muscle. Figures 19 to 22 are a series of drawings of sections through different levels of this fissular region.

Changes in the Region Between the Tympanic Opening of the Fissula and the Stapedial Joint.—In this region, namely, between the anterior margin of the oval window and the tympanic opening of the fissula, the cartilage is normally late in ossifying, and sometimes a margin of cartilage persists. However, sometimes in late fetal life this area undergoes ossification, usually forming trabecular bone of the intrachondrial type, as is shown between the stapes and the fissula in figure 36. Later these bony trabeculae can be surrounded by endochondral bone, as is shown in infant 93R (aged 14 months) in figures 37 and 38, or they can become reexcavated and partly refilled by



Figs. 39 to 46.—Photographs of sections through the fissular region, showing the cartilage or the osseous mass in the fissula blocking its tympanic opening. Figure 39 shows a section from embryo 45B (aged 29 [?] weeks); figure 40, from embryo 69 (aged 29 plus weeks); figure 41, from embryo 59 (aged 32 weeks); figure 42, from embryo 62 (aged 24 weeks); figure 43, from embryo 116A (aged 31 weeks); figure 44, from embryo 74 (aged 34+ weeks); figure 45, from infant 79 (aged 4 days), and figure 46, from infant 82 (aged 3 years).

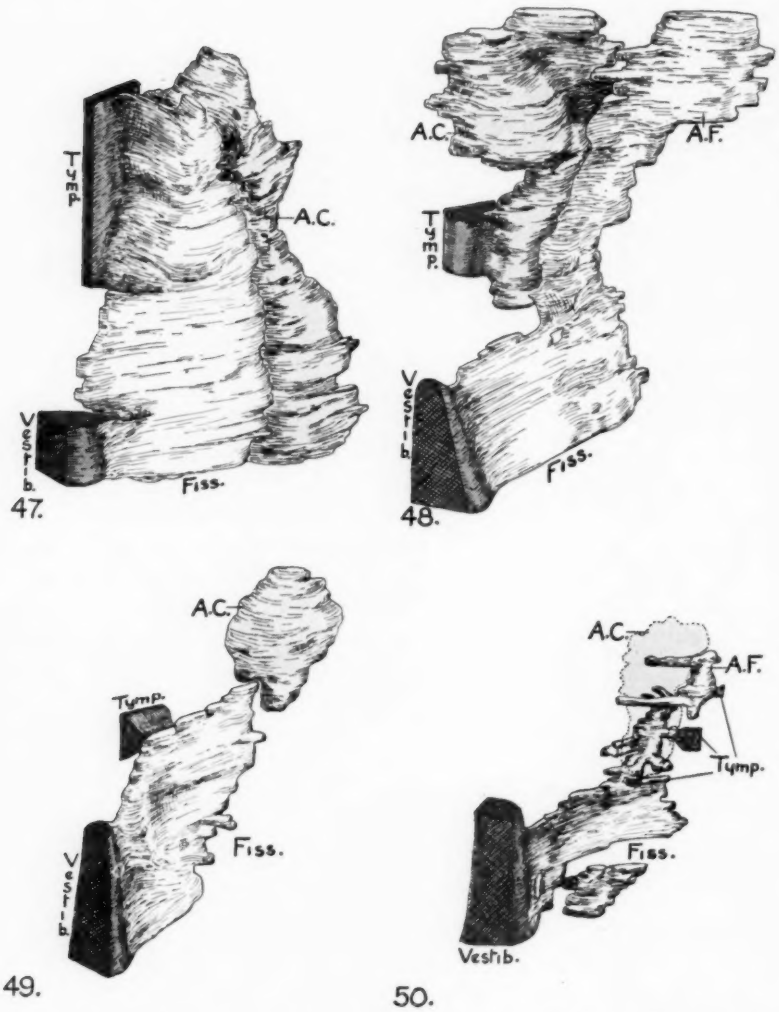
endochondral bone, as is shown in infant 79 (aged 3 days) in figure 45. As shown in the figures, this process may go beyond bounds and involve the fissula, either narrowing or obliterating it.

Changes at the Vestibular Opening of the Fissula.—This type is rare. There is only one specimen of this type in my collection (infant 121, aged 6 months); the vestibular end of the fissula is blocked with a mass of ossifying cartilage.

Comment.—As stated previously, the impression received from this study is that the mass of active cartilage arises from dormant cartilage at the margin of the fissula, for the purpose of reducing a large part of the fissula. Objections to this interpretation can be raised, for there are specimens as shown in figures 21, 22 and 48, in which the tympanic opening is plugged with active cartilage from the stapes to the semicanal for the tensor tympani muscle but the cartilage has not invaded the large aberrant upward extension of the fissula, although it lies adjacent to it. Then, too, it is difficult to explain why the formation of active cartilage in some instances is limited and in others involves the fissula generally. However, it is apparent that this activated cartilage appears at an embryologic age when ossification of the capsular cartilage should have been completed. Usually in the late months of fetal life the narrow rim of cartilage around the fissula is quiescent and has the appearance that it has in the adult. The cartilage mass, therefore, appears as a delayed attempt at further ossification. This active cartilage arising from the dormant cartilage at the margin of the fissula actually undergoes ossification at its margins later. The ossification may be either of the intrachondrial or of the osteoid type. The type formed may be dependent on the age of the subject when the cartilage mass forms. Thus, if the mass of active cartilage forms early, as in embryo 45B (180 mm. crown-rump length), which is when ossification of cartilage usually stops, then the usual formation of intrachondrial bone occurs at its periphery. This is shown in figure 39. If the process occurs later, as in embryo 116A (280 mm. crown-rump length), osteoid tissue may be formed (fig. 43). Later, such masses may be excavated by osteogenetic buds and filled with endochondral bone. The latter condition is shown in figure 45. In infant 83 (3 years old; fig. 46) there is a calcified cartilage mass which is surrounded by a mixture of intrachondrial bone, osteoid tissue and osteogenetic buds. This may represent a much delayed process.

In most cases, however, the osseous tissue formed is atypical or unstable. This has already been suggested and discussed by Wilson.[9]

9. Wilson, J. G.: Annual Report of the Otological Research Committee, Tr. Am. Otol. Soc., May 1935. Wilson.[8]



Figs. 47 to 50.—Drawings of models of the fissula ante fenestram. Figure 47 (left ear of embryo 70, aged 23 weeks), shows a mass of active cartilage (*A.C.*) along the cochlear border of the fissula. Figure 48 (left ear of embryo 62, aged 24 plus weeks) shows a mass of active cartilage (*A.C.*) in and above the tympanic opening. Figure 49 (left ear of embryo 45A, aged 29 weeks) shows a mass of cartilage filling the aberrant upward extension of the fissula. Figure 50 (right ear of infant 93R, aged 14 months) shows cartilage surrounding and reducing the fissula in the region of the tympanic opening and partly obliterating the aberrant upward extension. *Vestib.* indicates the vestibule; *Tymp.*, the tympanic cavity; *A.C.*, the mass of active cartilage; *Fiss.*, the fissula ante fenestram, and *A.F.*, the aberrant upward extension of the fissula ante fenestram.

PERCENTAGE OF EARS OF INFANTS AND FETUSES IN WHICH THESE CHANGES OCCUR

Tables 1 and 2 are designed to answer the third question and to indicate the number and percentage of ears (and the age of the subject) that show the specific type of change in the fissular region discussed

TABLE 1.—*The Types of Changes in the Fissular Region and the Number of Ears and the Age Group for Each Type*

	Human Fetuses				Infants		
Type of Fissular Region	Catalog No.*	Crown-Rump Length, Mm.	Approximate Age, Weeks	Sex	Catalog No.	Age	Sex
Fissula Open							
1. Normal	21	183	21	Male	101	4 mo. 26 da.	Female
	29A	190	21+	Female			
	7	205	23+				
	51	210	24	Male			
	46	222	25	Female			
	2	230	26				
	20	295	32+	Male			
	60	305	33+	Female			
	115	370	Term	Female			
2. Plug of cartilage or bone in fissula above tympanic opening	14	202	23	Male	93L	14 mo.	Male
	64	230	26	Female			
	42	240	27	Female			
	15	246	27+	Male			
	45A	260	29	Male			
	109	280	31	Male			
	66	290	32	Male			
	67	370	Term	Male			
	125	370	Term	Female			
3. Cartilage or bone between fissula and cochlea	29B	180	21+	Female	75	4 wk.	Female
	70	202	23	Female	103	1 yr. 10 mo.	Male
	114	230	26	Male	96L	3 yr.	Male
	116B	280	31	Female	96R	3 yr.	Male
4. New ossification around fissula from stapes to tympanic opening; fissula reduced and irregular	4	275	30+	.....	119	4 mo. 8 da.	Female
					106	5 mo. 6 da.	Female
					97	10 mo.	Male
					93R	14 mo.	Male
					81	6 yr.	Female
Fissula Blocked							
5. Cartilage in tympanic opening or in fissula nearby	45B	180	29 (?)	Male	79	5 da.	Female
	62	215	24+	Female	83	10 wk.	Male
	69	265	29+	Female	98	6 mo.	Male
	116A	280	31	Female	120	7 mo. 13 da.	Male
	59	290	32	Female	78	7½ mo.	Male
	68	310	34	Female	88	2½ yr.	
	74	315	34+	Male	82	3 yr.	Female
	95	...	Term	Male	118	5 yr.	
	102	...	Term	Male			
6. Ossified cartilage in vestibular opening of fissula					121	6 mo.	Female

* The letters A and B after catalog numbers indicate twins.

in the preceding paragraphs. It will be noted that the percentage of ears with an open fissula is greater among fetuses than among children. The fissula in 55 per cent of children is open. This percentage in children is higher than was suspected at first from casual observation. This false impression was due to the ears in group 4. In these ears there is considerable cartilaginous and bony reorganization involving the fissula, so that it is irregular and narrowed. From study of the individual sections the fissula appears to be blocked. However, a study

of the sections in series reveals that the fissula is continuous but irregular and much smaller. The five ears in this group originally considered as having blocked fissulae brings the percentage of open fissulae in children higher than the percentage of closed fissulae. At that, the percentage of open fissulae in fetuses is considerably higher than the percentage in children. This may be accounted for by the delayed processes of change which are not yet noted in fetal ears. Then, too, it is possible that some of the cartilaginous and bony changes do not reach their maximum development until after fetal life.

This statistical study raises a question which cannot be answered here: Why are almost one-half the fissulae of young children blocked? A statistical study of adult ears must be made to determine the significance of the observations. If the fissula is intact in most adult ears, then the question remains: Is the blocking of the fissula in children a temporary condition which is later corrected and is the fissula reestab-

TABLE 2.—*The Number and Percentage of Open and Closed Fissulae in Human Fetuses and in Infants*

	Human Fetuses		Infants		Total	
	Number of Ears	Percentage	Number of Ears	Percentage	Number of Ears	Percentage
Open fissulae	23	71.875	11	55	34	65.4
Closed fissulae	9	28.125	9	45	18	34.6

lished, or does the blocked fissula represent a pathologic condition that not only leads to death but causes the abnormalities of the fissula?

RELATION BETWEEN THESE CHANGES IN THE FISSULAR REGION AND THE FORMATION OF OTOSCLEROTIC BONE

In a discussion of the fourth question, may it be clear from the start that this is a consideration not of clinical otosclerosis but rather of localized patches of changed bone to which the term otosclerotic bone has been applied.

It is well known that such otosclerotic patches of bone are most commonly found in the otic capsule in the fissular region. The capsular changes noted in the fissular region in human fetuses and infants, therefore, arouse the suspicion that there may be a developmental relationship between them and the otosclerotic foci observed in older persons. A study of the ears of fetuses and infants leaves the distinct impression that the capsule in the fissular region is in an unstable state and that the cartilaginous or bony masses represent a delayed attempt at stabilization. A glance at later stages in this process shows that it has produced bone quite different from that found in the rest of the otic capsule. This is shown in figures 35, 37, 38, 45 and 46, in which

more or less definitely circumscribed areas of dark-stained unstable bony tissue can be seen. These areas are not otosclerotic bone, but it is possible, or even likely, that an attempt will later be made at reorganization, first by excavation or resorption and then by rebuilding to form otosclerotic bone. In figure 45 osteogenetic buds have already partly canalized the osseous mass at the stapes and in the fissular region and suggest early steps in the process of reorganization.

The series of ears on which these observations are based permits no positive conclusions, but observations strongly suggest that the cartilaginous or osseous mass which arises in the fissular region in a certain percentage of fetal and infant ears is a focus in which later reorganization may occur, leading to the formation of otosclerotic bone. A study of ears of older children and young adults should throw more light on this subject.

SUMMARY

1. In a certain percentage of ears of infants and fetuses a mass of active cartilage apparently arises from the dormant cartilage along the fissular margin. This mass later changes into atypical bone.

2. The cartilaginous or osseous mass occurs most frequently (1) along the cochlear margin of the fissula, (2) in the aberrant fissular extension above the tympanic opening, (3) in the fissula at or near its tympanic opening or (4) in the region where the fissula extends toward the stapedial joint.

3. The cartilaginous or osseous mass apparently occurs in or around an unusually large portion of the fissula and tends to reduce or even to obliterate parts of the fissula by its development.

4. Since the cartilaginous mass changes to atypical or unstable osseous tissue, it is possible that later an attempt is made to resorb and reorganize such an area, which may result in otosclerotic bone.

BRONCHOSCOPIC STUDY OF CARCINOMA OF THE LUNG

AN ANALYSIS OF THREE HUNDRED CASES OF BRONCHIAL CARCINOMA WITH ONE HUNDRED POSTMORTEM EXAMINATIONS

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The history of the progress in the more accurate understanding of disease of the chest runs pari passu with the more widespread use of bronchoscopy. The bronchoscope has proved to be of the utmost help not only in definitely establishing the existence of carcinoma in cases in which it is unsuspected or in confirming the clinical diagnosis of its presence but also, on occasion, in making it possible to negate a positive clinical diagnosis.

As experience was gained in the bronchoscopic examination of carcinoma of the lung it was realized that in a certain proportion of cases a definite diagnosis cannot be made by endoscopy. In fact, occasionally no abnormality of the bronchial tree can be detected, although the case ultimately proves to be one of carcinoma. In this group of cases the neoplasm cannot be visualized because of its peripheral location, and hence no pathologic tissue presents itself for biopsy. Pressure effects of the tumor or of metastatic glands nevertheless may be visible.

The only conclusive bronchoscopic evidence of carcinoma of the lung is the demonstration by histologic examination of a biopsy specimen removed with the aid of the endoscope. If endoscopic removal of tissue is not possible the diagnosis must be based on other histologic evidence, viz., microscopic examination of fluid from the chest and specimens obtained from lymph nodes, puncture of the lung or thoracotomy. We are convinced that certain abnormalities of the bronchial tree are suggestive of a malignant process, especially in association with evidence obtained from clinical examination and roentgenograms. However, we have repeatedly observed similar variations in nonmalignant pathologic conditions of the thoracic cage. Thus, freely bleeding mucosa has been frequently noted in the edematous mucous membrane proximal to a foreign body, so-called granulations, in a benign tumor,

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such as an angioma, and in venous engorgement associated with mediastinal compression due to lesions other than carcinoma. A diminution in caliber of the lumen of a bronchus may be the result of carcinomatous infiltration (a fact which can usually be demonstrated by punching out a small piece of tissue for biopsy); yet we have often found it associated with simple compression of the lung by pleural effusion or with the atelectasis associated with paralysis of the phrenic nerve. Narrowing of the lumen may be due to nonmalignant stricture or extrabronchial pressure from a mediastinal gland, as in Hodgkins' disease. Rigidity of the bronchial wall with distortion of the bronchial tree and limitation of the bronchial movements can be seen in tuberculosis, longstanding bronchiectasis or chronic pulmonary abscess.

Several problems presented themselves at the start of our investigation: 1. What percentage of bronchial carcinomas are so situated that a positive diagnosis can be made by the microscopic examination of bronchoscopically removed specimens? 2. How many are impossible of visualization and detection even by the most skilled bronchoscopist? 3. What are the gross bronchoscopic appearances of carcinoma of the bronchus? 4. To what extent can the bronchoscopist aid the surgeon in estimating the operability and in determining the type of operation to be used? The answer to these questions can best be found in a study of postmorten material in cases in which bronchoscopy was performed. For this purpose we analyzed 100 postmortem specimens of pulmonary carcinoma and compared the data obtained with the bronchoscopic findings noted during life. In addition, we reviewed the bronchoscopic findings in 200 proved cases of carcinoma of the lung in which necropsy was not performed. The analysis of these 300 cases which we studied follows.

AGE

Carcinoma of the lung is more prevalent in the fourth and fifth decades of life. The youngest patient in our series was a 19 year old Negro and the oldest a 76 year old white man. The lesion is much more frequent in males than in females, the ratio being about 3 to 1.

TABLE 1.—*Age Incidence of Carcinoma of the Lung*

Age, Years	Diagnosis Proved Post Mortem	Diagnosis Proved Clinically	Total
19–20	1	0	1
21–30	2	2	4
31–40	11	24	35
41–50	27	48	75
51–60	41	89	130
61–70	16	35	51
71–80	2	2	4
	100	200	300

LOBES INVOLVED

The upper lobe seems to be the site of predilection of carcinoma of the lung. This is particularly true of the upper lobe of the right lung, which is the most frequently involved region.

Primary carcinoma of the upper lobe of the lung in our postmortem material was noted in 48 per cent of cases, with 31 per cent occurring in the right lung. In our larger series, without autopsy, the neoplasm was considered primary in the upper lobe in only 38 per cent. The first figure, 48 per cent, is, of course, more accurate, since the original site of the growth was determined at autopsy. In the second series the bronchoscopist reported the lobe involved according to the location from which the specimen was taken. When no such biopsy specimen was available, the lobe involved was established roentgenographically. Often a neoplasm arising in the bronchus of the upper lobe infiltrates proxi-

TABLE 2.—*Lobes Involved by Carcinoma*

Lobes	Diagnosis Proved Post Mortem	Diagnosis Proved Clinically		Total
		Number	Percentage	
Right lung:				
Main bronchus	18	44	22.0	62
Upper lobe	31	49	24.5	80
Middle lobe	1	4	2.0	5
Lower lobe	12	22	11.0	34
Left lung:				
Main bronchus	8	25	12.5	33
Upper lobe	17	28	14.0	45
Lower lobe	13	28	14.0	41
	100	200		300

mally to involve the main stem bronchus, where it may first be seen by the bronchoscopist. Ordinarily the biopsy specimen is taken from this area, and the neoplasm is thought to arise in the main bronchus rather than in the branch in the upper lobe. For this reason our statistics in the second series may show a higher incidence of carcinoma of the main bronchus than really existed.

PATHOLOGY

Although we have used the terms carcinoma of the bronchus and carcinoma of the lung freely they are not strictly interchangeable. It would be more accurate to include both under the heading of carcinoma of the lung and perhaps refer to the large proportion which definitely arise in the bronchial mucosa as bronchiogenic. The vast majority of carcinomas, from 80 to 90 per cent, are truly bronchial in origin. However, it has not yet been definitely disproved that a small minority, so-called parenchymal growths, may not arise in the pulmonary alveoli.

It is beyond the scope of this paper to enter into any discussion as to the exact origin of primary carcinoma of the lung, and we do not

intend to include any complete description of its histopathologic picture. However, a comprehensive knowledge of the gross anatomic distribution is essential in explanation of the clinical syndrome and bronchoscopic findings.

We have adopted the topographic classification of pulmonary carcinoma suggested by Wessler and Rabin. Grossly, one may recognize three distinct groups, both from the pathologico-anatomic location in the bronchial tree and from the resultant clinical findings. These are carcinoma of a large bronchus (hilus) (62 per cent), carcinoma of a small branch bronchus (18 per cent) and parenchymal carcinoma (20 per cent).

Carcinoma of a Large Bronchus (Hilus).—This group comprises the majority of all bronchiogenic carcinomas. The neoplasm is to be found in the main bronchus, in the branch in the upper, middle or lower lobe or in the immediate terminal division of one of these branches. Actually, this includes tumors located in bronchi of the first, second and third orders. It is to be noted that the main stem bronchus before bifurcation into the upper and lower lobes is designated a bronchus of the first order. The exact site of origin of the lesion is found in most cases somewhere along the bronchial mucosa. From this point growth proceeds both by direct extension and through the lymphatic system. Local spread of the tumor soon results in either (1) obstruction of the bronchial lumen or (2) ulceration of its mucosa.

Carcinomatous stenosis of a bronchus due to direct invasion by neoplasm may occur in one of several ways: (*A*) Obturating stenosis. The obstruction results from the actual protrusion of the neoplastic growth per se into the lumen of the bronchus. (*B*) Infiltrating stenosis. The obstruction is produced by the submucous infiltration of the bronchial wall, causing a circular or funnel-shaped stenosis. The latter is a later stage in a submucosal malignant growth, which, originating in the mucosa of the bronchus, first produces a flat elevation of the mucosa. From this point the malignant process spreads submucosally through the lymphatic system, producing secondary changes in the mucous membrane. Because of this manner of spread, the overlying mucosa is thrown into folds and appears ruffled or dimpled. As this process continues more of the wall becomes infiltrated, and when the entire circumference is involved circular stenosis may result. Viewed through the bronchoscope, the lumen may appear funnel-shaped, the apex of the cone being at the original site of the lesion and the base toward the hilus, or it may produce a tubular stenosis, with no variation in diameter of the lumen at different levels, in which case bronchoscopic diagnosis is made possible only by observing the longitudinally placed folds in the mucosa. (*C*) Extrabronchial pressure. This form is usually associated

with marked distortion of the bronchial tree. Pressure may be due to glandular metastases or to encroachment by the malignant growth itself. The bronchial mucosa overlying the tumor may appear normal or may show signs of invasion by contiguity.

Carcinomatous ulceration is frequently associated with some degree of bronchial obstruction. Ulceration results either from the breaking down of the neoplasm or from secondary bacterial infection. The appearance is usually that of a fungating mass with rolled-up granular edges. The base of the ulcer rests on a deeply infiltrating growth. Carcinoma of a large bronchus is further characterized by very early metastases to the regional lymph nodes and distant parts of the body. Metastases from the original site may spread toward the hilus, producing large regional nodes, or to the periphery in the form of lymphangitic carcinosis.

Carcinoma of Small Branch Bronchi.—This group comprises from 15 to 20 per cent of all pulmonary carcinomas. The tumor is situated distal to the terminal division of the stem bronchus to the upper, middle or lower lobe. The lesion is therefore located in a bronchus of a lower order, namely, from the fourth to the ninth order. These neoplasms are truly bronchial in origin, although their distribution is in the peripheral part of the bronchial tree. One may recognize two distinct types among these carcinomas of small branch bronchi from both the clinical and the pathologic point of view.

1. Peripheral Localized Form: About 50 per cent of the carcinomas of small branch bronchi are to be found in the periphery of the lung, involving the bronchi of the smallest dimension. The growth usually remains localized at its site of origin for some time but may then show a marked tendency to infiltrate the chest wall and metastasize to the ribs. Regional nodes are seldom involved early in the course of the disease, and distant metastases develop only later as a rule. The method of spread is primarily toward the periphery of the lung, thereby sparing the larger bronchi.

2. Peripheral Infiltrative Form: The other 50 per cent of carcinomas of small branch bronchi are much more malignant and devastating. They metastasize early to the regional nodes and infiltrate along the lymphatic vessels toward the hilus. This infiltration is submucosal and produces a puckering of the mucosa, with an obliteration of normal bronchial markings. By this process circular stenosis of the larger bronchi may result. The metastatic nodes produce marked distortion of the bronchial tree and pleural effusion with or without paralysis of the recurrent nerve.

Parenchymal Carcinoma.—In this group are included the peripherally placed, localized, almost circumscribed neoplasms which probably

originate in the pulmonary alveoli. These parenchymal tumors comprise about 20 per cent of all pulmonary carcinomas.

The bronchial tree is in no place directly involved by the malignant growth. Even the minutest grossly visible bronchi are free from tumor infiltration at autopsy. The only abnormality of the bronchial tree that may be present is distortion of the lumen due to extrabronchial compression. This absence of bronchial involvement accounts for the paucity of pulmonary symptoms until the lesion is far advanced. Hemoptysis, pleural effusion and palsy of the recurrent nerve do not occur early in the disease. An important feature of these parenchymal growths is that they metastasize relatively late. Regional bronchial nodes are not involved until the lesion is far advanced, or they may be spared entirely. Proof of this has been obtained by repeated histologic examination of the nodes, no evidence of tumor being noted. At necropsy it is not uncommon to see a large broken-down peripheral carcinoma without any metastases.

Comment.—It should again be emphasized that all bronchiogenic carcinomas are infiltrative growths and as such produce rigidity and fixation of the bronchial wall. The tumor involves the bronchus over a fairly wide area and is always sessile. Pedunculated or polypoid growths have not been found to be carcinomatous and should not be included among bronchiogenic carcinomas. They are usually adenomas, strongly resembling adenocarcinomas, yet showing a different histologic picture and a nonmalignant clinical course. Rabin and Moolten have been able to separate these growths from true carcinomas. In our entire series of 300 cases there was not a single case in which a true carcinoma presented a pedunculated appearance. In the current literature there have appeared numerous communications on the excellent results obtained in the treatment of carcinoma with endoscopic removal, implantation of radium or direct cauterization. In all cases in which such treatment was beneficial, the growth was pedunculated, and the histologic appearance, as shown in photomicrographs, could definitely be recognized as characteristic of adenoma.

In 1930 one of us in an article on adenoma of the bronchus showed the true nature of these growths to be adenomatous and stressed their nonmalignant course. Such cases of adenoma as have been reported with successful results should not be permitted to go on record as representing cures of primary carcinoma of the lung.[1] We recently reviewed our experiences with 23 adenomas of the bronchus and differentiated them from carcinomas.

1. Kramer, R., and Som, M. L.: Ann. Otol., Rhin. & Laryng. **44**:861 (Sept.) 1935.

In reviewing our 100 pathologic specimens we were able to subdivide them definitely into the three groups, as previously outlined: 62 cases of carcinoma of a large bronchus (hilus), 18 cases of carcinoma of small branch bronchi and 20 cases of parenchymal carcinoma.

BRONCHOSCOPIC APPEARANCE

The endoscopic appearance of a bronchiogenic carcinoma may be variable within limits. The particular type of growth concerned and the duration of its existence are obviously paramount factors affecting the bronchoscopic picture. The presence or absence of superimposed infection may materially alter the view obtained. Huge mediastinal nodes, especially between the upper lobe and the trachea, occasionally obstruct the bronchial lumen and make visualization of the neoplasm impossible, even though it is in a main bronchus. Nevertheless, in spite of these disturbing features, the appearance of carcinomas of large or small bronchi is rather uniform.

The hilar carcinoma presents on bronchoscopic examination either stenosis or ulceration of the bronchus. Stenosis is much more frequent and may be either partial or complete, depending on the age of the lesion. It should be clearly emphasized that when there is an obstructing lesion only the most proximal part of the neoplasm permits of bronchoscopic examination. The area that can be seen through the bronchoscope usually comprises at most only a small portion of the entire neoplasm. As stated previously, stenosis may be obturative or infiltrative.

A. Obturative Stenosis.—The lesion itself protrudes into the lumen and is responsible for obstruction by reason of its own mass. The tumor is granular, friable and sessile.

B. Infiltrative Stenosis.—The obstruction is the result of infiltration into the bronchial wall, with subsequent narrowing of the lumen. The mucosa is replaced by flat tumor tissue, which is difficult to cut. The funnel-shaped stenosis represents the later stage of a submucous carcinoma. We have observed the earlier stage of this type of growth before stenosis supervened.

The earliest lesion of this variety of bronchiogenic carcinoma is often difficult to detect bronchoscopically and may be overlooked. The normal bronchial marking may be distorted over an isolated area. The rings seem to be drawn close together in this region. The mucosa presents a ruffled and wrinkled appearance. There is a slight, flat elevation, which represents the origin of the tumor, proximal to which the mucous membrane may be thrown into folds. The entire lesion may be only 1 cm. in diameter. We have observed a lesion of this size in a patient who presented few pulmonary signs and in whom roentgenographic examination of the chest revealed no abnormality. Biopsy of

the overlying mucosa, however, revealed an infiltrating squamous cell carcinoma. Later phases of this type of carcinoma are more frequently noted. The area involved is considerably larger. Normal bronchial markings are absent. The wall is infiltrated and rigid, and bronchial movements may be absent. The elevation is marked and involves more of the bronchial circumference. The mucosa appears granular and infiltrated, finally producing the tubular or funnel-shaped stenosis, which is more commonly seen. It should be borne in mind that whereas this appearance is most commonly associated with carcinoma of the lung, other pathologic conditions of the chest may present similar pictures. Final proof is dependent on the careful histologic examination of the biopsy specimen. Here again there is room for error, in that if the specimen is not taken from tumor tissue proper it will show no evidence of new growth. It is often advisable when doubt exists to repeat the bronchoscopy and obtain another specimen.

Ulceration.—Certain appearances of bronchial tumors are very suggestive of carcinoma. A fungating growth with an ulcerated surface, usually accompanied with some degree of stenosis, is almost invariably a carcinoma. Occasionally, cartilaginous rings are involved and lie exposed in the ulcerated bed.

Carcinoma of Small Branch Bronchi.—A neoplasm of this type, when visible, presents a uniform bronchoscopic appearance. However, not all these neoplasms are so located as to be detected endoscopically. In fact only 50 per cent of these neoplasms encroach far enough proximally to be within the range of vision of the bronchoscopist.

Viewed bronchoscopically, the tumor presents itself as an infiltrative growth. The bronchial walls seem rigid. The lesion is found in a fourth order bronchus, but in advanced stages the third order or even more proximally placed bronchi may be involved. The mucosa appears granular and ruffled, as if thickened. Frequently the spur between branch bronchi becomes thickened and shortened. In such cases the condition has been reported as normal by the bronchoscopist "except for slight thickening of the mucosa or of the spur between divisions of the bronchi." Pathologic reports subsequently establish the significance of these "slight thickenings" by demonstrating the presence of carcinomatous infiltration. Peripherally the bronchial lumen occasionally narrows and tapers to a point. For the sake of brevity we have designated this type of growth as infiltrative. Marked distortion of the bronchial tree is fairly frequently noted in these cases. The final diagnosis can be established only by biopsy of tissue.

A localized tumor of a branch bronchus cannot be seen endoscopically unless it has grown into a large bronchus, usually a preterminal event. The same is true of parenchymal growths, although distortion of the bronchial tree and rigidity of the walls are common.

CLASSIFICATION AS TO BRONCHOSCOPIC APPEARANCE

In tabulating the bronchoscopic observations in the histologically proved cases of bronchiogenic carcinoma we found certain features peculiar to the type of growth concerned. Thus, all neoplasms of large bronchi were associated either with some degree of bronchostenosis or with ulceration. In the few early cases of submucous hilar carcinoma in which we had the opportunity of making a bronchoscopic study the infiltration and thickening of the mucosa were the prominent features; the stenosis was less marked.

To facilitate the classification of the bronchoscopic appearances of carcinomas of the lung, we decided to group under individual headings only those findings that were constant: stenosis, ulceration and infiltration. When more than one or when all were present, the predominant feature was given preference.

In 70 per cent of the entire series of 100 cases biopsy showed the presence of a carcinomatous growth. Further study was made to determine the percentage of cases of the different types of carcinomatous growth and to determine the characteristic bronchoscopic appearance of each type.

Large Bronchus.—In 60, or 97 per cent, of the 62 cases comprising this group the biopsy specimen was carcinomatous. In 43, or 70 per cent, the dominant lesion was stenosis, while in the remaining 17, or 27 per cent, ulceration was more manifest. In 2, or 3 per cent, of the cases of carcinoma of a large bronchus a biopsy specimen showing carcinomatous growth was unobtainable. In both cases there were ample presumptive evidences of malignancy, but the actual lesion could not be directly visualized in either case. It seems advisable to review these 2 cases.

CASE 1.—N. K., aged 54, was examined bronchoscopically on Oct. 17, 1927. Paralysis of the right recurrent nerve was noted. The right side of the trachea was pushed toward the left, causing marked narrowing of the tracheal lumen. The spur at the bifurcation was thickened. Displacement was so marked that the bronchoscope could not be introduced into the main bronchus of the right lung. The postmortem examination on Jan. 10, 1928, revealed that the upper two thirds of the anterior surface of the right lung was covered by a coarsely granular, firm sheet of tumor tissue. In the upper portion of the mediastinum of the right lung, filling out the entire angle between the trachea and the upper lobe, was a large, firm mass, continuous with the sheet of tumor tissue. The tumor involved the apex also. The mass in the angle was 12 cm. in diameter and included several large lymph nodes, completely encasing the innominate vein, the right subclavian artery and the recurrent nerve. The main bronchus of the right lung presented a moderately coarse, nodular mucous membrane, which was firm on section and grayish white. The mucous membrane was markedly thickened, reaching its greatest width at the termination of the main bronchus.

Comment.—In this case the primary lesion was located in the main bronchus of the right lung but could not be visualized endoscopically because of the huge obstructing metastatic nodes.

CASE 2.—H. I., aged 19, was examined bronchoscopically in October 1933. Inspiratory stridor was marked. Paralysis of the left recurrent nerve was noted. The tracheal lumen was markedly narrowed by intense edema of the mucosa and was compressed from side to side by extrabronchial masses. In the right lung the main bronchus could not be entered because of the marked distortion of the bifurcation and the pressure on the lower end of the trachea. Further manipulation was deemed inadvisable for fear of inciting a reaction which might completely obliterate the already partially stenosed lumen. At postmortem examination huge metastatic nodes were present on the right side of the trachea, obstructing the entrance to the main bronchus of the right lung. The large mass of carcinomatous glands on the left completely encased the aorta and the recurrent nerve. The carcinoma was primary in the main bronchus of the right lung, invading toward the hilus.

Comment.—The primary lesion was unapproachable because the huge metastatic nodes prevented entrance into the main bronchus. The intense edema of the bronchial mucosa precluded further manipulation.

Small Branch Bronchi.—In 10, or 55 per cent, of the 18 cases in this group biopsy showed the presence of carcinomatous tissue. In 8 cases infiltration was noted bronchoscopically. In 2 cases examination was made late in the course of the disease, after invasion of a larger bronchus with resultant ulceration had taken place. In one of these cases a carcinomatous gland was responsible, whereas in the other the tumor perforated into the bronchial lumen.

In the remaining 8 cases, 44 per cent, the biopsy specimen was not carcinomatous. In 3 of these cases bronchoscopic examination revealed a bronchial tree without any abnormality. In the other 5 cases bronchial distortion and evidences of extrabronchial pressure were noted. A further analysis of these 8 cases seemed warranted. In 6 cases the postmortem examination demonstrated that the bronchi as far down as the fifth order were free from neoplastic growth. Microscopic examination of the bronchial wall substantiated this gross observation. The tumors were situated in the periphery of the lung and involved the terminal bronchi. In 5 of these cases there was extensive local infiltration of the chest wall with metastases to the ribs.

It is obvious that carcinoma of small branch bronchi, spreading in the manner just described, cannot be detected early by bronchoscopic examination. These growths have been designated in the topographic classification of Rabin and Neuhof [2] as localized peripherally. The bronchial lesion is far beyond the range of vision in the bronchial tree, and a definite diagnosis cannot be made on endoscopy. However, the local metastasis to the chest wall is usually apparent on the roentgenographic film.

2. Rabin, C. B., and Neuhof, H.: J. Thoracic Surg. **4**:147 (Dec.) 1934.

In the 2 cases, in which biopsy of suspicious-appearing, thick mucous membrane revealed no tumor tissue, necropsy demonstrated a tumor in the upper lobe of the left lung in both instances. The spurs between the terminal divisions of the bronchi in the upper lobe were thickened. The mucosa in the bronchi of the fourth order was irregular, and the tumor could be seen extending proximally from the bronchi of a lower order. Histologic examination of bronchi of the fourth order showed tumor cells in the walls and in the lymphatic vessels around the bronchi, infiltrating from peripheral branches. The diagnosis might have been confirmed if a more representative biopsy specimen had been obtained.

It must be borne in mind that the time interval between the biopsy and the postmortem examination is an important factor in the comparison of the two pictures obtained. Months may elapse following bronchoscopy before the patient dies. The lesion may then be far more extensive than it was at the time of the bronchoscopic examination. As a matter of fact, early carcinoma of small branch bronchi may not infiltrate proximally enough to permit of bronchoscopic visualization. Nevertheless, months later autopsy may reveal extension of the growth into the larger bronchi. The bronchoscopist cannot always be held to account for the apparent discrepancy in the observations.

Parenchyma.—In these 20 cases either no specimen was taken for biopsy or the biopsy tissue was not carcinomatous. In every instance the bronchi were free down to the terminal branches on postmortem examination. Even the closest scrutiny at necropsy failed to reveal any communication with the bronchial lumen. In 9 cases no abnormality of the bronchial tree had been revealed on endoscopy. In 10 cases extrabronchial pressure, with distortion of the bronchial lumen, had been reported and was substantiated post mortem. In 1 case considerable bronchial distortion was present post mortem without having been noted previously.

CLINICALLY PROVED CASES IN WHICH NO POSTMORTEM EXAMINATION WAS MADE

Of the 200 clinically diagnosed cases of pulmonary carcinoma in which no postmortem examination was made, 152, or 76 per cent, were definitely proved as such by the microscopic examination of a bronchoscopically removed biopsy specimen. In 48, or 24 per cent, a biopsy specimen showing carcinomatous growth was unobtainable.

Bronchoscopic examination revealed stenosis in 98 cases (49 per cent), ulceration in 31 (15.5 per cent) and infiltration in 23 (11.5 per cent; 8 cases, 4 per cent, of infiltration into the main bronchus)—a total of 152 cases (76 per cent) in which the biopsy specimen showed carcinomatous growth.

It is logical to assume, judging from our postmortem statistics, that all cases in which bronchostenosis is the dominant feature are cases of carcinoma of a large bronchus. Similarly, those in which ulceration is the predominant lesion are of this variety. A peripheral growth with invasion of the larger bronchi was present in 2 cases in our first series and might have been mistaken for a hilar carcinoma, thus proving an occasional exception. Thus, in 129, or 64.5 per cent, of the 152 proved cases the carcinoma was located in a large bronchus. Besides these there were 8 cases of involvement of a large bronchus which were included under the heading of infiltration, although the specimen was taken from the main bronchus in each case. These represent the earlier submucous type of growth, with an infiltrative appearance, unaccompanied by a marked degree of bronchostenosis. These were described bronchoscopically as showing thickened spurs, a nodular mucosa or flat elevations in the main bronchus. Sufficient submucosal infiltration to produce the so-called circular stenosis had not occurred as yet. The entire lesion was 1 cm. in diameter in 1 case, and the resultant stenosis was negligible. Roentgenographic examination of the chest in this instance revealed no abnormality in the lungs. This is the only instance in our series in which roentgenography revealed no abnormality, yet a bronchoscopically removed specimen showed the presence of a squamous cell carcinoma. Without the biopsy a diagnosis would otherwise have been impossible. We should like to emphasize the importance of the infiltrative appearance in the main bronchi for the early diagnosis of submucosal carcinoma. This infiltrative appearance, rather than the presence of a pedunculated growth, should be stressed in the bronchoscopic detection of early carcinoma. If the latter 8 cases are included with the 129 already described, this makes a total of 137 cases, or 68.5 per cent, of carcinoma of a large bronchus.

As demonstrated in our previous series, infiltration in the smaller bronchi is always due to carcinoma which has infiltrated proximally. We may regard our 15 cases as being of this type.

In 33 of the 48 cases in which endoscopic examination or study of a biopsy specimen removed endoscopically did not show evidence of carcinomatous growth the bronchial tree showed no abnormality, while in 15 there was evidence of distortion and extrabronchial pressure. Although examination of endoscopically removed tissue did not confirm the clinical impression in these 48 cases, the final diagnosis was established by other means. The histologic diagnosis in 26, or 55 per cent, of these cases was obtained by biopsy of axillary or cervical nodes (in 17 cases), by microscopic examination of fluid from the chest—the Mandelbaum technic (in 7 cases) or by puncture of the lung (in 2 cases). In the remaining 22 the diagnosis rested on clinical and roentgenographic evidence. In 9 cases there were metastases to the brain and to bones.

In the other cases hemoptysis, clubbing of the fingers or paralysis of the recurrent nerve, together with roentgenographic evidence, was noted.

When the growth is not visualized bronchoscopically and autopsy is not performed, one is not always certain as to the type of growth present in any case. However, the clinical syndrome is usually typical, and the roentgenograms are fairly characteristic for the various groups.

Of the 48 nonproved cases of carcinoma, 5 can be definitely classified as cases of carcinoma of a large bronchus clinically associated with bronchostenosis and atelectasis. The diagnosis was confirmed by microscopic examination of specimens from metastatic nodes. In these cases a biopsy specimen was not obtained endoscopically because tremendous adenopathy prevented the proper visualization of the bronchial tree. Sixteen of the 43 remaining cases can be regarded as cases of parenchymal tumor on the basis of the roentgenograms and the clinical picture. In the remaining 27 cases the tumors most probably were situated in small branch bronchi.

ASSOCIATED POSTMORTEM OBSERVATIONS IN ONE HUNDRED CASES

Esophagus.—Necropsy revealed carcinomatous infiltration of the esophageal wall in 8 cases in this series, although dysphagia was present in only 2, in which actual perforation of the esophageal wall with invasion of tumor tissue into the esophageal lumen had occurred. Both patients had previously been examined esophagoscopically following bronchoscopy, and specimens had been removed from cauliflower-like intrusions into the esophageal lumen. The pathologic diagnosis based on these specimens as well as on the specimens removed bronchoscopically was squamous cell carcinoma. At postmortem examination fungating masses were observed eroding the anterior wall of the esophagus and partially obstructing its lumen. The primary neoplasm in both cases was located in the main bronchus of the right lung. As the growth was a squamous cell carcinoma, it is barely possible that it was primary in the esophagus in these cases. It should be noted that in 6 cases in which there was infiltration of the esophageal wall by carcinoma there were no symptoms referable to the esophagus.

Tuberculosis.—An associated tuberculous cavity was observed in 4 cases at autopsy. In 1 case tuberculous infection involved the upper lobe of the left lung, whereas the bronchiogenic carcinoma was located in the upper lobe of the right lung. In 2 instances the neoplasm seemed intimately connected with the wall of the cavity. The sputum showed acid-fast bacilli in only 2 of these cases.

One is not justified in drawing any conclusions as to the etiologic relationship between tuberculous cavitation and bronchiogenic carcinoma. Tuberculous cavitation is noted at routine postmortem examinations in

about 4 per cent of cases. Our results seem to militate strongly against the concept of the etiologic rôle of tuberculous infection in carcinoma of the lung.

Frank tuberculosis of the bronchial wall was not observed post mortem in this series. The tentative bronchoscopic diagnosis of tuberculous ulceration of the bronchus was made in 3 cases, but in each instance necropsy disproved the diagnosis. Twice the bronchoscopist reported finding "small, round ulcerations, from 3 to 5 cm. in diameter, with a flat, yellowish base surrounded by an areola of congestion, not elevated above the adjacent mucous membrane." Marked distortion of the bronchial tree was noted. Smears were taken, but acid-fast bacilli could not be demonstrated. At autopsy carcinoma infiltrating a small branch bronchus was present, with no evidence of tuberculous infection. The flat ulcerations were the result of lymphangitic infiltration of the submucosa in 1 case and invasion from a metastatic gland in another. In the third case several whitish plaques were observed on the mesial wall of the main bronchus and in the upper lobe of the right lung, with evidences of extrabronchial pressure. The impression gained by a bronchoscopist was that of a healed tuberculous ulcer with tuberculous adenopathy. Paralysis of the left recurrent nerve was similarly explained. Necropsy disclosed carcinoma of a large bronchus, but no evidence of tuberculous infection.

Biopsy specimens were not taken in these cases, first, because the appearance seemed to be that ordinarily associated with tuberculous ulceration and, second, for fear of initiating spread of the infection by manipulation. On the other hand, in 2 cases in which carcinomatous stenosis was suspected healed tuberculous strictures were noted post mortem.

Syphilis.—The Wassermann reaction of the blood was 4 + in 3 cases and 3 + in 1 case. It was negative in all other cases. Syphilis of the lung was not present in a single case. Again, there was no apparent relationship between syphilis and carcinoma in this series.

Suppurative Disease of the Lung.—Suppurative disease of the lung is a frequent complication of bronchiogenic carcinoma. Pulmonary abscess, bronchiectasis and empyema are the most common suppurative processes encountered. Areas of pneumonitis so often seen surrounding the tumor mass proper and recurrent pneumonitis affecting an entire lobe are most apt to be limited and are not strictly included under this heading. Surgical intervention is never indicated in a case of pneumonitis. Pulmonary suppuration may be disclosed at autopsy in cases in which it was clinically unsuspected.

Pulmonary Abscess.—In the cases under discussion the essential lesion was always a carcinoma, with the suppurative process secondarily

ingrafted. The infection was never the initial event. We may thus more correctly refer to the condition in our cases as secondary pulmonary abscess or suppurative disease of the lung with cavitation. Secondary pulmonary abscess was noted in 14 of the 100 cases in which postmortem examinations were made.

In 6 instances the infection was putrid, and a clinical diagnosis of pulmonary abscess had been made. At endoscopy a bronchiogenic carcinoma was detected, which was responsible for the anaerobic infection distally. In 3 of these 6 cases the wall of the cavity was seen post mortem to be infiltrated by a malignant growth, whereas in 2 cases infiltration could not be demonstrated in the pyogenic membrane. In 1 case of putrid infection of the lung the bronchoscopically removed specimen showed no evidence of tumor, but necropsy revealed carcinoma in small branch bronchi.

In the remaining 8 cases cavitation of the lung was the result of the breaking down of a peripherally placed carcinoma, with the necrotic tumor tissue forming the core. Anaerobic infection and foul odor were not present.

Suppurative Bronchiectasis.—It is well known that bronchial occlusion dependent either on obstructive growth within the lumen or on extrabronchial pressure may result in atelectasis, bronchiectasis or secondary pulmonary abscess.

Obstruction of the main bronchus is likely to produce diffuse inflammation of the bronchial tree, with bronchiectasis rather than secondary pulmonary abscess. The latter more frequently is the result of bronchostenosis in the bronchi of lower orders. Carcinoma of a large bronchus is particularly prone to result in secondary pulmonary infection with bronchiectasis. Bronchoscopically, profuse purulent secretion emanating from a partially stenotic large bronchus is therefore very common. When the instrument can be passed beyond the obstructive lesion, bronchial dilatation, thinning of the bronchial septums and other stigmas of bronchiectasis may be visualized. At autopsy an entire lobe may be observed to be the seat of a huge bronchiectatic cavity.

Suppurative Pleurisy.—The pleural cavity may be infected as the result of (1) spread from necrotic, infected tumor tissue, (2) rupture of a secondary putrid pulmonary abscess, (3) suppurative pneumonitis or (4) secondary infection of malignant effusion. In these cases a bronchopulmonary fistula may be present.

MODIFIED BRONCHOSCOPIC APPEARANCE OF CARCINOMA DUE TO SUPERIMPOSED INFECTION

The usual endoscopic appearance of a putrid pulmonary abscess, the characteristic foul odor and the recurrent flow of anaerobic pus from

an acutely inflamed bronchial orifice, are well known. The edematous mucosa which narrows the lumen of the affected bronchus may be only slightly altered by the presence of an infiltrating growth. Gross differentiation is often impossible, and only microscopic examination of specimens will demonstrate the presence of a tumor. It is therefore wise to make a biopsy of all abnormal tissue.

When the bronchial tree seems distorted and there are signs of extrabronchial pressure in addition to those of primary pulmonary abscess, one should be suspicious of pulmonary neoplasm. In 5 instances of bronchiogenic carcinoma complicated by pulmonary abscess distortion was prominent. Distortion due to primary abscess of the lung per se is relatively rare and was observed only once in a series of 100 consecutive cases of putrid pulmonary abscess.

Patients with recurrent suppurative infection of the pleural cavity of obscure etiology should be examined bronchoscopically. In 4 of our cases thoracotomy was performed for empyema without a previous endoscopic examination. Because the wound did not heal and infection persisted, bronchoscopic examination was resorted to, and only then was the diagnosis of bronchiogenic carcinoma established.

PARALYSIS OF THE RECURRENT AND PHRENIC NERVES

Paralysis of the phrenic nerve was noted in 9 cases. It occurred 6 times on the left side and 3 times on the right. In 1 instance paralysis of the right phrenic nerve was the result of pressure by a metastatic node due to a primary carcinoma in the main bronchus of the left lung. Paralysis of the phrenic nerve is much more frequently the result of malignant disease than of tuberculosis.

Paralysis of the recurrent nerve was present in 14 cases. It occurred 5 times on the right side and 8 times on the left. In 1 case both nerves were involved. At autopsy gross anatomic dissections of the recurrent nerves threw considerable light on the subject of this type of paralysis.

In 3 cases paralysis of the left recurrent nerve was associated with a bronchogenic carcinoma on the right side. The primary tumor was in the upper lobe of the right lung in 2 instances and in the right main bronchus in the other case. The left nerve was involved by a metastatic gland on the side opposite the original tumor.

In 1 case paralysis of the right recurrent nerve was the result of a carcinomatous gland from a tumor in the lower lobe of the left lung. In another case metastasis to the right lobe of the thyroid gland from a primary growth in the lower lobe of the left lung exerted pressure on the right recurrent nerve, with resultant palsy. In still another case the left recurrent nerve could be dissected in its entire course from its entrance into the larynx to its origin from the vagus nerve under the arch of the aorta. Carcinomatous involvement or undue pressure was

nowhere to be noted. However, the left vagus nerve was entirely encased in carcinoma. Incidentally, the patient had tachycardia. Huge nodes on either side of the trachea accounted for the only case of paralysis of both recurrent nerves.

Although many observers have noted its occurrence with tuberculosis, we have not had a single proved instance of paralysis of the recurrent nerve due to tuberculosis. In 3 cases paralysis of the recurrent nerve was accounted for clinically by tuberculosis of the lung with adenopathy and pressure. The subsequent course revealed the presence of carcinoma of the lung in each case. In 2 cases this was proved by bronchoscopic examination, and in 1 case, by postmortem examination. We are of the belief that paralysis of the recurrent nerve never can be attributed to tuberculous infection. Malignant growth is by far the most common etiologic factor. Paralysis of the recurrent nerve in a case of obscure disease of the chest is hence highly suggestive of carcinoma.

PLEURAL EFFUSION

Pleural effusion in sufficient amount to be detected clinically and roentgenographically was present in 27 cases. The routine histologic examination of fluid from the chest by the Mandelbaum technic for tumor cells gave positive evidence of malignancy in 16 cases, or 60 per cent. Since a bronchoscopic examination had been made in all these cases, a comparison of the findings is of interest.

In 9 cases a histologic examination of a bronchoscopically removed specimen and of fluid aspirated from the chest showed the presence of tumor cells. In 7 cases the biopsy specimen was not cancerous, but tumor cells were demonstrated in fluid from the chest. In 11 cases the fluid did not contain tumor cells, but the biopsy specimen showed malignant change. These observations demonstrate that, even in the presence of a pleural effusion associated with carcinoma, a positive diagnosis can be more frequently made by means of study of a bronchoscopically removed specimen than by examination of fluid from the chest.

BRONCHOSCOPY AS AN AID TO THE SURGEON

The bronchoscopist can aid the surgeon in determining the extent and type of operation necessary in carcinoma of the lung in two ways: (1) by establishing the absence of invasion of the larger bronchi in cases of parenchymal carcinoma and in some cases of peripheral bronchial carcinoma and (2) by determining the uppermost limits of involvement of the larger bronchi, so that the surgeon may know whether lobectomy or pneumonectomy should be attempted. In the event that the mucosal invasion is between the orifice of the upper lobe and the tracheal carina, an operation is futile.

SUMMARY

A bronchoscopic examination was made in 300 cases of pulmonary carcinoma. In 222 cases, or 74 per cent, the diagnosis was proved by the histologic examination of a bronchoscopically removed biopsy specimen.

A comparison of the antemortem bronchoscopic appearance and the clinical manifestations with the postmortem observations has led us to adopt the topographic classification of pulmonary carcinoma into (1) carcinoma of a large bronchus, (2) carcinoma of branch bronchi and (3) parenchymal carcinoma.

The bronchoscopic observations and the associated clinical and pathologic phenomena associated with each of these types have been described.

We feel that an understanding of the value and of the limitations of bronchoscopy, as here delineated, will be of aid to the bronchoscopist, to the clinician and to the surgeon.

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A REFINEMENT OF THE EXTERNAL FRONTO-ETHMOSPHENOID OPERATION

A NEW NASOFRONTAL PEDICLE FLAP

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When an external operation for the treatment of suppuration of the frontal sinus fails it is usually for one of two reasons. Either the opening into the nose becomes inadequate or disease is left in supra-orbital ethmoid extensions with continuance of symptoms.

In 1898 Lothrop [1] reported a study of the surgical anatomy of the frontal sinuses. Almost everything that has since been written on the subject can be found in this paper. He pointed out that the full extent of the nasal portion of the floor of the frontal sinus is not used because of thick bone anteriorly and medially. In 1914 [2] he read a paper describing an operation he had used with satisfactory results in many cases. The method, reported more fully later,[3] provided a large opening into the nose by removing the interfrontal septum, all the nasal portion of the floor of both frontal sinuses and the upper part of the anterior portion of the nasal septum. To obtain this large opening he utilized the floor of a healthy sinus in cases of unilateral disease. The heavy bone between the sinus and the nose was removed with a burr. The sinus was entered through the front wall, and care was taken to leave a supporting shell of nasal bone and the ascending process of the maxilla to prevent the soft tissue from falling in to narrow the opening. He contended that the failure of operations based on Jansen's [4] technic introduced in 1893 was due to this obstruction of the opening. He probably was right.

However, only by removing the floor of the frontal sinus in its orbital portion and sacrificing the lacrimal bone and the os planum can the supra-orbital ethmoid extensions be dealt with. Lothrop's operation eliminates one cause of failure but leaves the other. He advocated

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1. Lothrop, Howard A.: Anatomy and Surgery of the Frontal Sinuses and Anterior Ethmoidal Cells, Ann. Surg. **28**:611 and 747, 1898; **29**:75 and 135, 1899.

2. Lothrop, Howard A.: Ann. Surg. **59**:937, 1914.

3. Lothrop, Howard A.: Frontal Sinus Suppuration with Results of New Operative Procedure, J. A. M. A. **65**:153 (July 10) 1915.

4. Jansen, A.: Arch. f. Laryng. u. Rhin. **1**:135, 1893.

preliminary intranasal midturbinectomy and ethmoidectomy as the first stage of his procedure. It is now known that this operation is seldom adequate.

Since the work of Jansen, procedures which satisfy the requirements in regard to the extensions of the ethmoid cells have been developed by Knapp,[5] Killian,[6] Lynch,[7] Sewall [8] and Smith.[9] There are still failures due to the cause stressed by Lothrop. Granulations from the soft tissues on the orbital side meet those arising from the cancellous bone on the medial side of the surgical nasofrontal opening, and eventually this opening is narrowed by a scar. The trauma produced by attempts to dilate the scar results only in the formation of more scar.

In 1934 Sewall [10] described a pedicle flap of mucous membrane to line the medial side of the nasofrontal opening and prevent the bridging by granulations. It has been used successfully in several cases. When it is properly applied both the causes of failure in the operation on the frontal sinus are avoided. There are cases, though, of large frontal sinuses with narrow nasal vaults, and particularly of those with bilateral involvement, in which the large opening made possible only by Lothrop's plan is desirable. In these cases the medial flap cannot be employed because the central bony structures are removed.

For these conditions I have devised a flap which is swung laterally. It employs the mucosa of the septum. The experience of Sewall and me has demonstrated that an operative nasofrontal opening remains patent if one side is epitheliated. The flap accomplishes this and avoids the first cause of failure without depending on the bony support stressed by Lothrop. The bone may, therefore, be removed to provide access to the ethmoid and sphenoid sinuses, according to the usual technic of Sewall and others. The second cause of failure may thus be avoided. Because the approach is at this level, the work on the bone may be done with the usual instruments. The operation is also safer than that described by Lothrop, because of better exposure of the danger area. This wider exposure makes it possible to work over to the opposite side with one incision in cases in which the ethmoid and frontal sinuses of the other side are healthy, but it is deemed advisable to utilize the entire nasal extent of the floor of the frontal sinus. A portion of the floor of the healthy frontal sinus may be denuded, and a flap from the septum on

5. Knapp, A. H.: Arch. Ophth. **28**:50, 1899.

6. Killian, G.: Arch. f. Laryng. u. Rhin. **13**:59, 1902.

7. Lynch, R. C.: Laryngoscope **31**:1 (Jan.) 1921.

8. Sewall, E. C.: External Operation on the Ethmosphenoid-Frontal Group of Sinuses Under Local Anesthesia, Arch. Otolaryng. **4**:377 (Nov.) 1926.

9. Smith, Ferris: Management of Chronic Sinus Disease, Arch. Otolaryng. **19**:157 (Feb.) 1934.

10. Sewall, E. C.: Ann. Otol., Rhin. & Laryng. **44**:307 (June) 1935.

the same side may be swung up to cover it. Cases of disease of a medial frontal cell or suppuration of a pneumatized crista galli communicating with the frontal sinus fall in the class in which this procedure is indicated.

OPERATION

The local anesthesia, incision of the skin, elevation of the periosteum, etc., are carried out as described by Sewall. A horizontal groove is chiseled through the nasal bone and ascending process of the maxilla at the lacrimal level of the middle of the fossa. Care is taken not to injure the underlying nasal mucosa. The frontal sinus is entered at the most medial portion of the orbital floor, and this opening is joined to each end of the horizontal cut. The pyramid of bone thus outlined is fractured and removed. The nasal mucosa is seen arching over the vault of the nose and down on the septum. It is not disturbed. The ethmoid vessels are ligated or coagulated and cut, and the ethmoid mass is removed, the sphenoid sinus opened, etc., as described elsewhere. The entire orbital extent of the floor of the frontal sinus is then removed. Particular care is given to any supra-orbital ethmoid extensions. All bony ridges must be smoothed. In large frontal sinuses the removal of the floor is completed laterally through a counter incision in the outer portion of the eyebrow. The front wall, as well as the floor, of these lateral extensions is removed, and the cavity is kept packed until obliterated by granulations. There is little deformity.

In cases of bilateral involvement the same procedure is then carried out on the opposite side, although the operation can be performed as a second stage on the fifth or sixth postoperative day.

The next step is the fashioning of the flap. The periosteal surface of the nasal mucosa is seen in the lower part of the anterior portion of the bony opening, forming the vault of the nose (fig. 1). It is easy to separate it from above downward from the perpendicular plate of the ethmoid bone and septal cartilage. The elevation is continued for about 2 or 3 cm. into the nose and anterioposteriorly for as wide a strip as the anatomic structure permits—usually from about 1 to 1.5 cm. The limits are the cribriform plate posteriorly and the root of the nose anteriorly.

A horizontal incision is made at the lower edge of the elevated portion of the septal mucosa, and vertical incisions are carried up to the vault from each end of this horizontal cut, a flap measuring about 1.5 by 4 cm. thus being freed, which is attached laterally to the ascending process of the maxilla (fig. 2).

The interfrontal partition is then entirely removed, and the heavy bone in the midline is attacked from above. This bone consists of the nasal process of the frontal bone and the thick upper ends of the nasal

bones and the ascending process of the maxilla, with which it articulates. A shell of bone is left to support the root of the nose. The denuded portion of the perpendicular plate is easily removed from above downward. In cases of unilateral involvement all this may be done from one side, and when the perpendicular plate of the ethmoid bone is removed the mucosa of the opposite side of the septum is seen. This can be fashioned into a flap for the unopened side, as suggested. In cases of

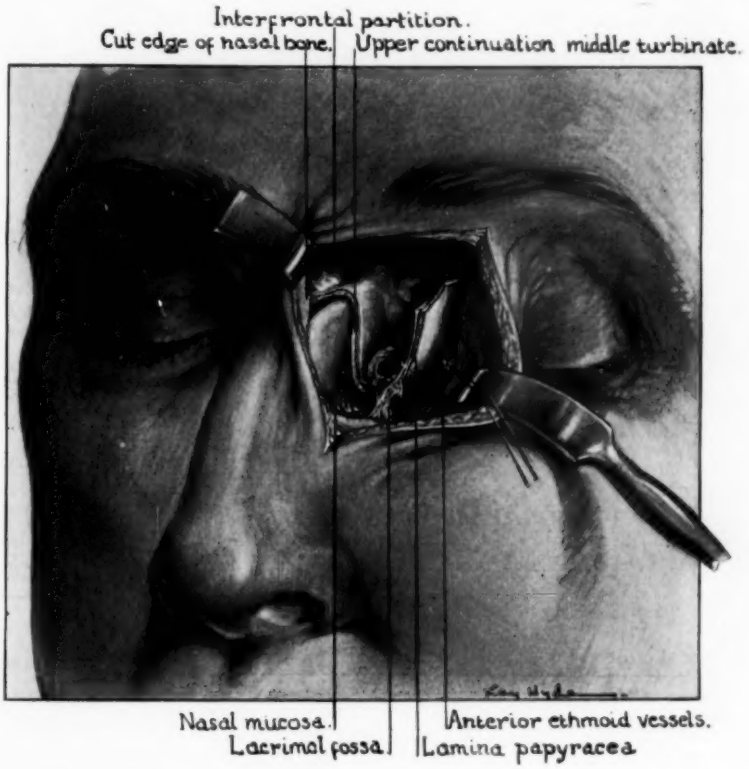

Fig. 1.—Drawing showing the periosteal surface of the nasal mucosa in the lower part of the anterior portion of the bony opening, forming the vault of the nose.

bilateral involvement some of the work on the bone is done from each side, but if the procedure is to be carried out in two stages it is probably best to remove the denuded portion of the perpendicular plate at the first operation.

The flaps are unfolded to extend over the floor of the frontal sinus and are held in place against the orbital periosteum by lengths of gall-bladder tubing 3/8 inch (0.95 cm.) in diameter passed through the nos-

trils and into the corresponding frontal sinuses. The incisions in the skin are closed with clips. No dressings are applied. The clips are removed on the third day, and the tubes on the fifth day or later.

The end-result is the conversion of both frontal sinuses and the upper part of the nose anteriorly into one cavity, lined on each side by mucosa, continuous with that over the ascending process of the maxilla. This mucosa extends over the orbit to line the floor. Epithe-

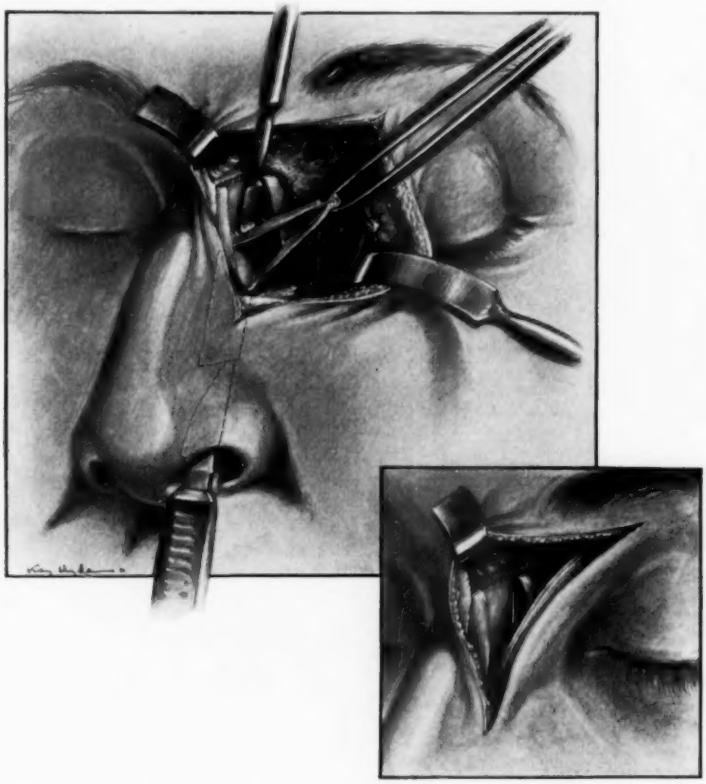

Fig 2.—Drawings showing the fashioning of the flap. The upper figure illustrates elevation of the mucosa from the septum, and the lower, the flap unfolded to lie against the orbital periosteum.

lium spreads from the flap and from the healthy sinus (in cases of unilateral involvement) to cover the healthy granulations that fill in from the periphery of a sinus which has been stripped of its mucosa. Small sinuses close to the point of obliteration, but the granulations remain healthy as long as there is free drainage into the nose. The flap insures this.

These pedicle flaps of mucous membrane were worked out by Sewall and me to solve a real difficulty. Then the article of Smith [9] came to our attention, in which he mentioned the use of a muscosal flap by Bárány. A search of the literature revealed that in 1924 Bárány [11] arrived at the same solution as Sewall. He did not obtain as large a flap, because he did not elevate the mucosa below the incision into the nose (through the ascending process), but the flap he used was essentially the same. He described another type of flap using mucosa of the frontal sinus, but I do not believe that the mucosa of a diseased sinus should be saved. At the end of his article he mentioned that Sourdille had been making independent use of a similar flap for years but had not described it in the literature. Sourdille made a larger flap, which was swung laterally. In 1930 he published his technic.[12]. It differed from mine in that he did not utilize septal mucosa. Lothrop [2] described the use of a pedicle graft of skin (from the upper lid). Smith utilized a free skin graft. Others have proposed the wearing of a rubber tube for a long time, and still others, the permanent installation of a silver prosthesis in the nasofrontal opening.

That the problem exists is well attested by the number of solutions, and it is not surprising that the same answer has been worked out independently by several surgeons.

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11. Bárány, R.: Acta oto-laryng. **7**:41, 1924.
12. Sourdille, Maurice: Ann. d. mal. de l'oreille, du larynx **49**:10, 1930.

ESOPHAGITIS

II. A PATHOLOGIC AND CLINICAL STUDY

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Wilks and Moxon [1] in 1889 gave a good pathologic description of inflammatory lesions of the esophagus. More recent textbooks, by Adami and Nicholls,[2] Kaufmann and Reimann [3] (1929) and Bassler [4] (1922) have given detailed descriptions of the various types of esophagitis. According to these writers, simple acute catarrhal esophagitis is rarely seen at necropsy. The pathologic process consists in exfoliation of the superficial epithelium and congestion of the mucosa. Chronic catarrhal esophagitis is described as occurring most commonly in alcoholic addicts and persons with pulmonary disease. According to these workers, esophagitis is the result of repeated congestion brought on by other diseases. Wood [5] (1932) even stated that it may be a factor in the production of muscular hypertrophy, with narrowing of the lumen of the esophagus.

Follicular esophagitis is a name often used to describe a process in which the mucous glands participate markedly in the inflammation. The ducts are frequently blocked by secretion, and small retention cysts are formed. If these become infected, suppuration, erosion and ulceration may occur. The esophageal glands are liable to infection and are probably the chief route of infection from within.

True diphtheritic membranous esophagitis is very rare and, according to the literature, usually fatal. Clerf [6] stated in 1926 that stenosis

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Abridgment of a thesis submitted by Dr. Butt to the faculty of the Graduate School of the University of Minnesota in partial fulfilment of the requirements for the degree of Master of Science in Medicine.

1. Wilks, Samuel, and Moxon, W., quoted by Cushing.[51]

2. Adami, J. G., and Nicholls, A. G.: The Principles of Pathology: Systemic Pathology, Philadelphia, Lea & Febiger, 1909, vol. 2, p. 399.

3. Kaufmann, Edward, and Reimann, S. P.: Pathology for Students and Practitioners, Philadelphia, P. Blakiston's Son & Co., 1929, vol. 1, p. 629.

4. Bassler, Anthony: Diseases of the Stomach and Upper Alimentary Tract, Philadelphia, F. A. Davis Company, 1922, p. 492.

5. Wood, D. A.: Primary Idiopathic Muscular Hypertrophy of the Esophagus with Narrowing of the Lumen, Arch. Path. **14**:766 (Dec.) 1932.

6. Clerf, L. H.: Laryngeal and Esophageal Sequelae of Diphtheria, Laryngoscope **36**:912 (Dec.) 1926.

may occur as a result of deep membranous involvement. In smallpox a so-called pustular esophagitis has been described in which exfoliation and formation of ulcer frequently occur.

Another rare type of esophagitis is known as exfoliative esophagitis. Adami and Nicholls described it as being characterized by the desquamation of the lining epithelium in large flakes or even as complete cylinders. The etiology is unknown but the condition usually occurs in neurotic persons. Jacob [7] in 1916 reported the case of a neurotic patient who vomited an esophageal cast that was 11 inches (28 cm.) long.

Phlegmonous esophagitis is described as a purulent cellulitis which begins in the submucous coat of the esophagus. Raw [8] in 1901 described a case in which the condition began as a dissecting esophagitis. The patient vomited a cast of the esophagus, 8.5 inches (21 cm.) long. This was followed by complete stenosis. It was observed at necropsy that the process had extended to the external muscular coat. This type of esophagitis may result by direct continuation from the pharynx.

There are certain specific types of esophagitis that deserve special mention; these include esophagitis due to tuberculosis, syphilis, actinomycosis, blastomycosis and Oidium albicans.

Tuberculous esophagitis is nearly always secondary to advanced tuberculosis in other parts of the body. Flexner [9] (1893) found reports of only 17 histologically proved cases in the literature. Vinson and Dobson [10] in 1927 reported a case of tuberculous esophageal stricture in which the patient had extensive pulmonary tuberculosis. Torek,[11] however, in 1931 reported the case of a patient without active tuberculosis elsewhere. According to most observers, the esophageal involvement is due to extension of the disease from the peritracheal lymph nodes, larynx or pharynx. The ulceration is usually superficial, with smooth or irregular bases and thickened borders. In our series of cases we did not encounter an instance of tuberculous esophagitis.

Syphilitic esophagitis is not seen frequently, and, according to Abel,[12] only 1 or 2 of every 1,000 patients presenting themselves because of an

7. Jacob, F. H.: An Unusual Case of Esophageal Cast, Lancet **1**:998 (May 13) 1916.

8. Raw, Nathan: Membranous Oesophagitis: Expulsion of a Complete Cast of the Oesophagus, Lancet **1**:26 (Jan. 5) 1901.

9. Flexner, Simon: Tuberculosis of the Oesophagus (Larvae of Diptera in the Ulcers), Bull. Johns Hopkins Hosp. **4**:4 (Jan.-Feb.) 1893.

10. Vinson, P. P., and Dobson, H. E.: Tuberculous Stricture of the Oesophagus: Report of a Case, Am. Rev. Tuberc. **16**:53 (July) 1927.

11. Torek, F.: Tuberculosis of the Oesophagus: Report of Case Without Active Tuberculosis Elsewhere, Ann. Surg. **94**:794 (Oct.) 1931.

12. Abel, A. L.: Syphilis of the Oesophagus, with a Report of Two Cases, Lancet **2**:441 (Sept. 1) 1928.

esophageal lesion show this type of esophagitis. Wile [13] (1914), Myerson [14] (1927) and Abel have all given excellent descriptions of syphilitic esophagitis. The pathologic picture, according to these writers, depends on the stage of the disease. The condition apparently occurs in all patients with syphilis which has been allowed to progress to the secondary stage. It may give symptoms at this stage, but it is more commonly observed in the tertiary period. Usually it is present in the form of localized gummas in the upper or lower third of the esophagus. No instance of syphilitic esophagitis was noted in our series.

Esophagitis caused by actinomycosis or blastomycosis is very rare. Wade and Bel [15] in a review of the literature in 1916 stated that the case of systemic blastomycosis reported by Shepherd and Rhea [16] in 1911 was the only one on record in which the esophagus was involved. Vinson, Broders and Montgomery,[17] however, reported a case in 1928 in which the condition was primary in the esophagus. In our series we observed no case of esophagomycosis which was due to these fungi.

Oidium albicans, or thrush, is usually considered as occurring in poorly nourished children and in adults with cachexia or prolonged disease. More often it extends from the pharynx or mouth, but it may arise independently. In our series Oidium albicans occurred in 5, or 2.5 per cent, of the total numer of cases of esophagitis. It occurred in 1 infant 2 months of age and in 4 adults. All 5 patients had suffered from comparatively prolonged diseases and were in a debilitated state before death. The gross appearance of the esophagus varied. In 2 cases the mucous membrane in the part involved appeared grossly to be covered with long white, lustrous bands of pseudomembrane with a velvety feel. In the 3 other cases the pseudomembrane was yellow and semigranular. The entire mucous membrane was involved in 2 cases, the lower third in 2 cases and the upper third in 1 case. Microscopically the mycelia were clearly seen in sections stained by the Brown-Gram method. An excellent article by Bertha Fineman [18] in 1921 gave the

13. Wile, U. J.: Syphilis of the Esophagus, Am. J. M. Sc. **148**:180 (Aug.) 1914.

14. Myerson, M. C.: Syphilis of the Esophagus, M. Clin. North America **10**: 919 (Jan.) 1927.

15. Wade, H. W., and Bel, G. S.: A Critical Consideration of Systemic Blastomycosis, with Notes on Certain Special Features and Report of Five Cases, Arch. Int. Med. **18**:103 (July) 1916.

16. Shepherd, F. J., and Rhea, L. I.: A Fatal Case of Blastomycosis, J. Cutan. Dis. **29**:588 (Nov.) 1911.

17. Vinson, P. P.; Broders, A. C., and Montgomery, Hamilton: Blastomycosis of the Oesophagus: Report of a Case, Surg., Gynec. & Obst. **46**:255 (Feb.) 1928.

18. Fineman, Bertha C.: A Study of the Thrush Parasite, J. Infect. Dis. **28**: 185, 1921.

detailed microscopic description of these mycelia. The pseudomembrane formed by this growth seemed to attach itself to the epithelial lining. In a few places it was seen to overlie an ulcerated area, but apparently the fungi did not penetrate into the esophageal wall. Bacteria and cellular débris were scattered throughout this membrane. In none of these cases was the mouth infected with Oidium albicans.

Leukoplakia.—Leukoplakia of the esophagus was first described by Bucher [19] in 1893. Lindemann [20] in 1908 noted that leukoplakia was rather commonly found with esophageal varices and chronic esophagitis. Adami and McCrae [21] (1914) noted it commonly in chronic drunkards and concluded that it is caused by a low grade chronic inflammation which leads to epithelial proliferation. Sharp,[22] in his excellent review of the subject in 1931, said that it is commonly seen at necropsy. Cleland [23] (1934), however, noted only 1 instance of leukoplakic patches in 3,000 necropsies.

Most investigators assume that chronic irritation is the best hypothesis as to the etiology of this condition. It is apparently, in itself, symptomless unless it causes obstruction. It occurs, according to Sharp, more frequently in the lower third of the esophagus. Lindemann noted that in chronic esophagitis and in leukoplakia the thickness of the epithelium is often the same. The thickening in chronic esophagitis, however, is due to edema and that in leukoplakia to an actual increase in the number of cells. Aschoff [24] (1923) observed that the patches of leukoplakia are more resistant to postmortem changes. Occasionally, he noted giant cells beneath these patches. Sharp concluded that carcinoma of the esophagus is frequently associated with leukoplakia and that the leukoplakia may be a precancerous lesion similar to that in the oral cavity.

There were 31 cases (14.5 per cent) in our series in which a gross diagnosis of leukoplakia was made. The average age of the patients was 47 years. Grossly, the areas of raised epithelium appeared to be more resistant to postmortem processes than the normal mucous mem-

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19. Bucher, Robert: Beiträge zur Lehre von Carcinom zur Casuistik und Beurtheilung der multiplen Carcinom, Beitr. z. path. Anat. u. z. allg. Path. **14**:71, 1893.

20. Lindemann, August: Zur Pathologie der menschlichen Oesophagusschleimhaut, Virchows Arch. f. path. Anat. **193**:258 (Aug.) 1908.

21. Adami, J. G., and McCrae, John: A Textbook of Pathology for Students of Medicine, Philadelphia, Lea & Febiger, 1914, p. 624.

22. Sharp, G. S.: Leukoplakia of the Esophagus, Am. J. Cancer (supp.) **15**: 2029 (July) 1931.

23. Cleland, J. B.: Pathological Lesions Met with in the Oesophagus During Post Mortem Examinations, M. J. Australia **1**:588 (May 5) 1934.

24. Aschoff, Ludwig: Pathologische Anatomie: Ein Lehrbuch für Studierende und Aerzte, ed. 6, Jena, Gustav Fischer, 1923.

brane. Leukoplakia, however, did not occur more often in cases in which chronic esophagitis was present than in those in which it was not. In no section did the condition appear to be precancerous, and in no case was there a history of alcoholism. This condition of the esophagus is usually seen in persons who have been confined to bed for a long time. Usually, during this period they have not received a normal amount of solid and semisolid foodstuffs. The squamous epithelium of the esophagus therefore was not undergoing its usual desquamation, and the cells continued to pile up. This explanation has practically no proof, but it does appear reasonable that the increased thickness of the epithelial lining might result from this process.

MATERIAL EXAMINED

The material examined consisted of postmortem specimens from patients who had been studied at the Mayo Clinic and who had died because of a medical or surgical condition. The esophagi, obtained over a period of six years and four months, were examined grossly and microscopically. During this period 3,032 complete necropsies were made. The clinical data were obtained from the histories and hospital records in the files of the clinic.

PATHOLOGIC STUDY

A diagnosis of esophagitis had been made for 213 patients, or 7.02 per cent of the total of 3,032 patients. All final diagnoses were made by microscopic examination. In most instances the gross diagnosis was confirmed by microscopic examination of the tissue. In a small number of cases, however, a condition which was given a gross diagnosis of esophagitis was proved microscopically to be due only to postmortem digestion. Figure 1 shows the increase in incidence of esophagitis during the period of six years and four months.

In 11 cases (5.1 per cent) the esophagus was grossly dilated, and in 32 (15 per cent) the involved area was bile-stained. The sections of the esophagus most frequently involved were as follows: the lower third in 160 cases (75.2 per cent), the middle third in 5 (2.3 per cent), the upper third in 5 (2.3 per cent), the lower half in 17 (7.9 per cent) and the entire esophagus in 26 (12.9 per cent).

The type and appearance of the gross lesions varied greatly. Most often the ulcerations were superficial and were arranged in linear streaks along the long axis of the tube. Often there were seen numerous irregular superficial ulcerations (fig. 2*b*) with hemorrhagic or bile-stained bases. Frequently the normal esophageal folds were not present, and the entire mucosa was denuded. Less often the ulcers were oval and discrete (fig. 2*a*) and extended down to the layer of deep muscle. In a few instances the entire mucous membrane was covered with a

black coating (fig. 3*b*), as though it had been burned with an escharotic. Sometimes fresh bleeding points were observed in or near the ulcers. In 19 cases (8.9 per cent) a pseudomembrane was present. It varied in extent and color. In a few instances it covered the entire mucosa of the esophagus (fig. 3*a*), and occasionally it was present only in patches. It was white, gray, yellow, black or bile-stained. Not infrequently the mucous membrane appeared ulcerated or digested, and scattered about were a number of discrete, raised, shiny white patches. These were areas of thickened epithelium. In 2 cases a carcinoma had infiltrated the esophagus and had resulted in ulceration.

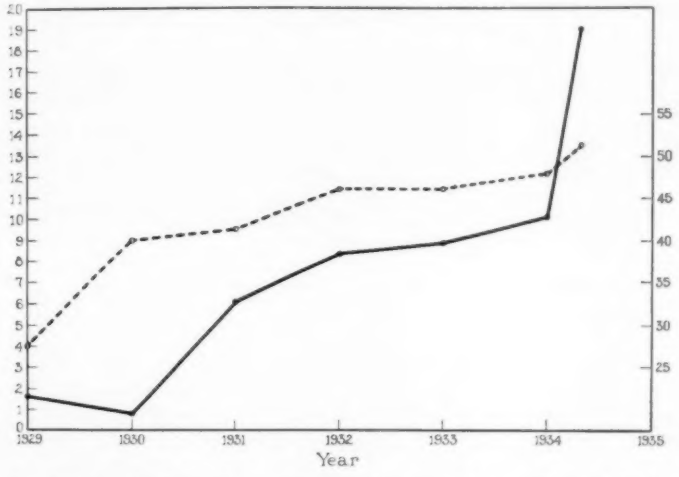

Fig. 1.—Chart showing the parallelism between the number of patients with esophagitis and the number of patients who vomited or were subject to removal of the gastric contents by tube. The ordinates at the left and the solid line indicate the percentage of patients with esophagitis. The ordinates at the right and the dotted line indicate the percentage of patients with esophagitis who had vomited or who had had the gastric contents removed by tube.

The gross pictures presented in this paper offer a more comprehensive description of the lesions commonly seen than one can hope to give verbally.

Microscopically the lesions were classified into three main types. The gross appearance often gave an indication as to the probable microscopic picture; but this was not uniformly true, since postmortem changes and changes due to embalming often altered the true picture of the lesion.

Acute Ulcerative Esophagitis.—In 163 (76.5 per cent) of the 213 cases a diagnosis of acute ulcerative esophagitis was made. This type of ulceration was characterized by a denuding of the lining epithelium.

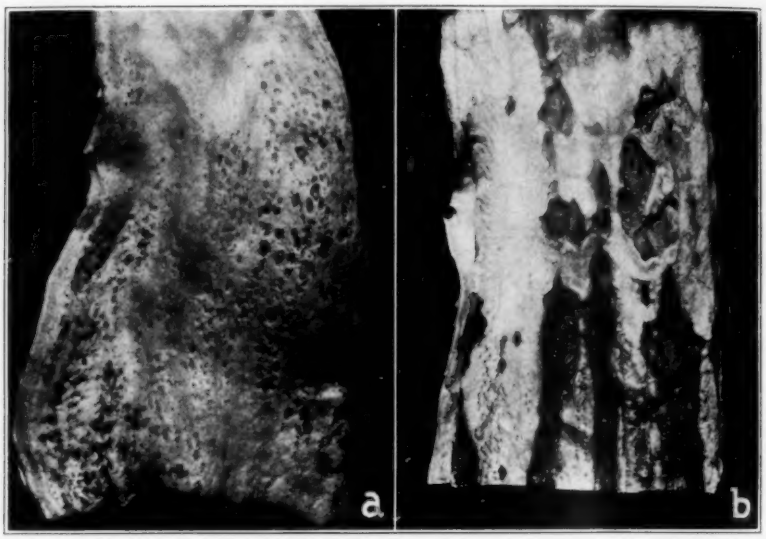

Fig. 2.—*a*, an esophagus showing multiple, fine discrete ulcers; *b*, the lower third of the esophagus, showing large discrete and confluent ulcers.

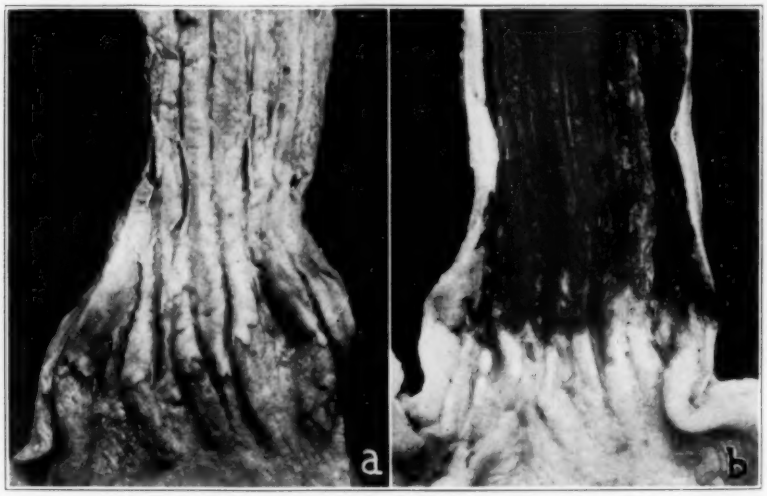

Fig. 3.—*a*, an esophagus showing diffuse pseudomembrane; *b*, an esophagus showing an ulcerated area that is deeply bile-stained.

The muscularis mucosa was often destroyed or infiltrated with an inflammatory cellular exudate. The predominating cells were polymorphonuclears. At times this inflammatory reaction was mild; yet again it was severe and involved the submucosa and both layers of muscle. This inflammatory process did not differ from other acute ulcerating processes elsewhere in the body. Often there was hemorrhage into the various coats and occasionally into the squamous epithelium itself. The mucous glands were frequently involved in the process, and the ducts of these glands were blocked and dilated. In the submucosa the vessels were often dilated and engorged with erythrocytes. Occasionally a ruptured vessel was seen emptying into the lumen of the esophagus. Thrombosed

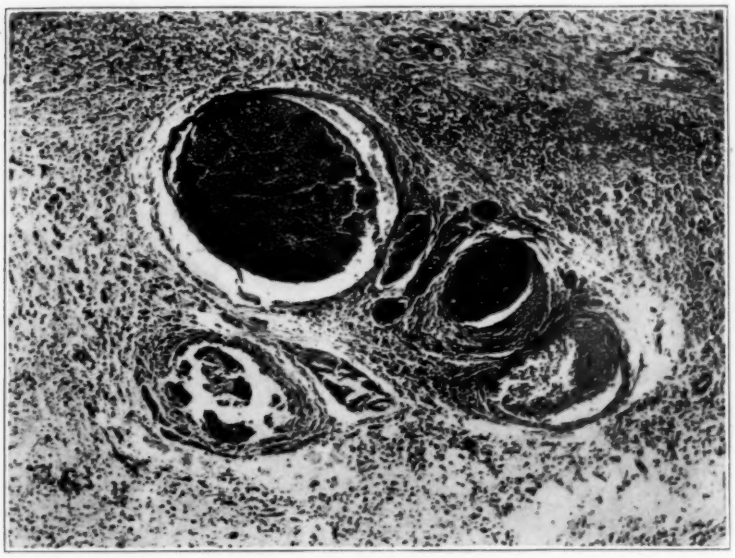

Fig. 4.—Thrombosed vessels in the wall of the esophagus surrounded by infected tissue (hematoxylin and eosin stain; × 90).

vessels were sometimes seen (fig. 4), and occasionally dilated lymphatic vessels filled with leukocytes were noted in the area involved.

A pseudomembrane was observed in 17 of the cases of acute ulcerative esophagitis. This membrane consisted of cellular and fibrinous débris and was usually bile-stained. As a rule it replaced the denuded squamous layer but often extended over the normal epithelium that remained. Numerous bacteria were occasionally observed in this membrane.

In 2 of the cases of acute ulcerative esophagitis periesophagitis was present in the outer fibrous coat. Here the infection had gone on to the formation of abscesses.

Subacute Ulcerative Esophagitis.—There were 31 cases (14.5 per cent) in this series of subacute ulcerative esophagitis. This type of inflammation was characterized by a denuding of the epithelial layer and an infiltration with leukocytes. There were an occasional polymorphonuclear, a few plasma cells, lymphocytes and eosinophils and beginning fibroblastic proliferation. In 2 cases a pseudomembrane was present, and in several there were marked congestion and hemorrhage into the esophageal coats.

Chronic Esophagitis.—There were 14 cases (6.5 per cent) of chronic esophagitis. The sections showed the usual picture of chronic inflammation. Many lymphocytes were present, and fibrous tissue had replaced most of the muscularis mucosa and submucosa. A number of sections revealed a marked increase in the thickness of the subepithelial connective tissue. The epithelial lining had apparently been denuded, and there were areas where the epithelium had attempted to grow back on this fibrotic base. In a few of these sections the muscularis mucosa appeared to be hypertrophied. The sections in which the subepithelial tissue increased probably represented the early stages of scar formation.

Such areas of fibrosis undoubtedly represent the result of repeated attacks of esophagitis with healing. The esophageal mucous membrane, owing to increased fibrosis, has a decreased blood supply, and its normal resistance is therefore decreased. It is easily conceived that such an esophagus would be a likely site for recurrent attacks of esophagitis.

THE RELATION OF ESOPHAGITIS TO BENIGN ESOPHAGEAL STRICTURE

Barclay [25] pointed out in 1913 that any inflammatory reaction in or around the esophagus may result in esophageal stricture.

Vinson [26] in 1923 reported 186 cases of benign stricture of the esophagus; in 18 (9.6 per cent) the etiology was unknown. Vinson [27] in 1931 reported 243 cases of benign stricture of the esophagus, in 40 (16.4 per cent) of which the etiology was unknown. This increase in the relative number of patients who have benign stricture of the esophagus of unknown origin is probably attributable to the decrease in incidence of esophageal stricture due to lye. More than 50 per cent of the strictures of unknown etiology were in the lower third of the esophagus. Only a few of the patients with such stricture had had any symptoms preceding the onset of dysphagia, and most of them had not experienced pain or a substernal burning after the onset of the

25. Barclay, A. E.: The Stomach and Oesophagus: A Radiographic Study, New York, The Macmillan Company, 1913, pp. 11 and 14.

26. Vinson, P. P.: Stricture of the Esophagus Occurring During Pregnancy, Am. J. Obst. & Gynec. **6**:346 (Sept.) 1923.

27. Vinson, P. P.: Cicatricial (Benign) Stricture of the Oesophagus: Tabulated Report of One Hundred and Eighty-Six Cases, Ann. Otol., Rhin. & Laryng. **52**:955, 1931.

illness. Vinson said that since the cardinal symptoms of peptic ulcer of the esophagus were not present, it was unlikely that peptic ulcer was a common etiologic factor in these strictures. Nevertheless, he stated that esophageal ulceration occurs more frequently than is generally recognized. He concluded that since the ulceration is usually superficial, healing does not, apparently, reduce the lumen of the tube enough to produce dysphagia. In this regard, the question that arises is how many times this ulceration with healing must occur before clinical symptoms of stricture are produced.

Vinson [28] (1921 and 1923) has reported a total of 9 cases of esophageal stricture that occurred following vomiting of pregnancy. In this condition one is certain that vomiting is the cause of the ulceration that produces the stricture. This again stresses the fact that fibrosis does follow the healing of esophageal lesions. Mussey and Randall,[29] writing in Curtis' book on obstetrics and gynecology (1933), gave anorexia and dysphagia as symptoms of vomiting of pregnancy. Esophagitis is perhaps frequently an accompaniment of all vomiting of pregnancy, but it rarely is of such severity that stricture results.

In our series the majority of the lesions were in the lower third of the esophagus. This is also the usual location of benign strictures of unknown etiology. On microscopic examination one can see healing and proliferation of fibrous tissue in a number of these (fig. 5). It seems reasonable to suppose, therefore, that if this process is repeated a number of times the resulting scar formation may be sufficient to produce a stricture.

Mosher [30] in 1933 stated:

> In other words, it is extremely common in chronic infection to find an increase of the subepithelial connective tissue of the esophagus. These findings, plus the fact that periesophagitis, hemorrhage, and hemorrhage into the musculature of the esophagus occur, give, I feel, abundant cause for webs or strictures of the esophagus at any part of its course. It becomes unnecessary, therefore, to rely on spasm, especially at the lower end of the esophagus, as the chief cause of esophageal obstruction.

CLINICAL STUDY

In this study the clinical histories and postmortem observations were carefully investigated in an effort to determine the possible relationship between esophagitis and the various associated diseases. Each condition

28. Vinson, P. P.: Oesophageal Stricture Following the Vomiting of Pregnancy, Surg., Gynec. & Obst. **33**:412 (Oct.) 1921; footnote 27.

29. Mussey, R. D., and Randall, L. M.: Toxemias of Pregnancy, in Curtis, A. H.: Obstetrics and Gynecology, Philadelphia, W. B. Saunders Company, 1933, vol. 1, p. 1006.

30. Mosher, H. P.: Involvement of the Esophagus in Acute and in Chronic Infection, Arch. Otolaryng. **18**:563 (Nov.) 1933.

was studied separately, but it is to be remembered that in many of the cases several factors were present, any one of which might have caused the esophagitis.

Vomiting and Removal of Gastric Contents by Tube.—Esophagitis is frequently the accompaniment of diseases in which there is frequent vomiting. Tileston[31] (1906) and Sheehan[32] (1920) observed that esophageal ulceration was favored by stenosis of the pylorus or duodenum or by an hour-glass stomach. Tileston noted pyloric or duodenal stenosis in 23 per cent of his 40 cases.

Smith and Lewald[33] (1915) commented on the frequency with which regurgitation accompanies colic in children. They stated that

Fig. 5.—Increase of the subepithelial connective tissue. This esophagus is undergoing healing, and the epithelium is attempting to grow on the fibrotic base (hematoxylin and eosin stain; × 140).

it is probable that the waves causing this regurgitation arise in the overactive part of the bowel.

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31. Tileston, Wilder: Peptic Ulcer of the Oesophagus, Am. J. M. Sc. **132**: 240 (Aug.) 1906.

32. Sheehan, J. E.: Ulcer of the Esophagus from the Standpoint of an Esophagoscopist: Its Cause, Symptomology, Diagnosis and Treatment, M. Rec. **97**:319 (Feb. 21) 1920.

33. Smith, C. H., and Lewald, L. T.: The Influence of Posture on Digestion in Infancy, Am. J. Dis. Child. **9**:261 (April) 1915.

Carlson, Boyd and Pearcy [34] (1922) observed experimentally that stimulation of the gallbladder, urinary bladder or intestine leads to esophageal activity. Burnham [35] (1923) observed roentgenographically in man a type of regurgitation similar to that observed in cats by Cannon.[36] It occurred most commonly in cases of disease of the gallbladder. Alvarez [37] (1928) expressed the opinion that stimuli to the vomiting center may stream constantly from a pregnant uterus, a dilated heart or a diseased gallbladder.

Table 1 indicates the apparent importance of vomiting and trauma in the etiology of esophagitis. Figure 1 shows the increase since 1929 in the number of cases of vomiting and the use of the stomach tube. This increase has paralleled somewhat the increase in the incidence of esophagitis. Table 1 shows that 46 per cent of all the patients had vomited or had had the gastric contents removed or both.

TABLE 1.—*Data on the Vomiting, the Removal of Gastric Contents by Tube and Dilatation in Two Hundred and Thirteen Cases of Esophagitis*

	Cases	Percentage
Vomiting	74	34.7
Removal of gastric contents by tube	64	31.4
Vomiting or removal of gastric contents by tube, or both	98	46.0
Vomiting, frequent or continuous	44	20.6
Removal of gastric contents by tube, frequent or continuous	45	21.1
Dilatation of stomach	28	13.1

Dilatation of the stomach apparently plays a minor rôle, but it is of some importance. The cardia, under the increased pressure produced by this condition, probably allows a more frequent flow of gastric contents into the esophagus.

Since esophagitis has been considered by a number of investigators to be an agonal or postmortem condition brought on by digestion of the mucous membrane of the esophagus by the gastric contents, it seemed important to determine, if possible, the acidity and enzymic activity just at and after death. One hundred postmortem specimens of gastric juice were examined for free and total acid contents by the Töpfer

34. Carlson, A. J.; Boyd, T. E., and Pearcy, J. F.: Studies on the Visceral Sensory Nervous System: XIV. The Reflex Control of the Cardia and Lower Esophagus in Mammals, Arch. Int. Med. **30**:409 (Oct.) 1922.

35. Burnham, M. P.: Importance of Indirect Roentgen Findings in Chronic Infection of the Biliary Ducts and Gallbladder, Am. J. Roentgenol. **10**:105 (Feb.) 1923.

36. Cannon, W. B.: The Movements of the Stomach Studied by Means of the Roentgen Rays, Am. J. Physiol. **1**:359, 1898.

37. Alvarez, W. C.: The Mechanics of the Digestive Tract: An Introduction to Gastroenterology, New York, Paul B. Hoeber, Inc., ed. 2, 1928, pp. 87, 288, 295 and 333.

gastric titration method (table 2). In an effort to obtain some determinations of the gastric pepsin, the gastric contents were secured immediately after death in a few instances. The determinations were made by Osterberg according to the method described by Vanzant, Osterberg, Alvarez and Rivers [38] in 1933.

Tables 2 and 3 show that free hydrochloric acid is not usually present in the stomach immediately after death. When it is present after death, it disappears rapidly. The acid-secreting power appears to be inhibited a few hours before death. In only 2 of the 8 cases was the presence

TABLE 2.—*Postmortem Gastric Analysis*

Hours Between Death and Necropsy	Cases	Free Hydrochloric Acid	Average Total Acid
1 to 3	52	4	32
3 to 6	13	2	28
6 to 9	12	0	29
9 to 12	18	0	39.3
12 to 15	4	0	17.5
15 to 18	1	0	7
Total	100	6	

TABLE 3.—*Analysis of Gastric Contents Removed Immediately After Death*

Case	Free Hydrochloric Acid	Total Acid	Pepsin, Units
1	0	8	100
2	0	4	84
3	0	28	1 to 1,080
4	0	12	0
5	6	24	1 to 980
5	0	20	0
6	0	38	0*
7	22	34	2,000
7	0	36	0†
8	0	10	0

* Postmortem examination was performed one and three-fourths hours after death.
† Postmortem examination was performed eleven hours after death.

of free hydrochloric acid demonstrated when the gastric contents were secured immediately after death. In 3 of these 8 cases studied for peptic activity the reading was greater than the normal maximum of 500 units. In only 1 instance was there sufficient acid to allow for optimal peptic activity.

It appears, then, that during the so-called agonal stage neither the acid nor the pepsin is of sufficient amount to produce severe irritation. Esophagitis probably begins in most cases some time before death.

38. Vanzant, Frances R.; Osterberg, A. E.; Alvarez, W. C., and Rivers, A. B.: Studies of Gastric Pepsin: Secretion of Pepsin in Cases of Duodenal Ulcer and Pseudo-Ulcer, J. Clin. Investigation **12**:557 (May) 1933.

It is clearly shown in table 4 that, regardless of what other factors one considers in the etiology (infection, nerve stimulation and so forth), vomiting and the use of the stomach tube hold first place.

The Rôle of Infection.—Not only can infection reach the esophagus by way of the blood stream, but the esophagus is almost constantly being bathed with organisms from the oral cavity. The anatomic structure of the esophagus, with its mucous glands opening free on the epithelial surface, makes it vulnerable to the extrinsic mode of infection. Frequently the dilated ducts of these glands are seen to be surrounded by an increased number of lymphocytes and polymorphonuclears, which indicates the presence of some infection. No doubt these ducts are often blocked, and infection leads to the formation of small or large abscesses. The lymphatic drainage of the esophagus makes it a possible target for infections of the abdominal cavity and its organs.

TABLE 4.—*Conditions Other Than Esophagitis Encountered in the Cases Studied*

Condition	Cases	Percentage of Total	Vomiting or Removal of Gastric Contents by Tube or Both
Peptic ulcer	42	20.1	50
Uremia	48	22.5	50
Chronic cholecystitis	50	23.4	48
Peritonitis	76	35.6	47.4
Hyperglycemia	9	4.2	44.4
Pneumonia	109	51.1	42.2
Hyperbilirubinemia	12	5.6	41.6
Chronic ulcerative colitis	11	5.1	18.0
Septicemia	18	8.4	16.6
Involvement of central nervous system	24	11.2	12.5
Average			37.7

Jackson,[39] in reviewing his cases of peptic ulcer of the esophagus, observed that there was evidence of chronic foci of infection in 90 per cent. Mosher[40] (1931) expressed the belief that the esophagus can be infected even before birth. He observed, further, that esophagitis of the terminal portion of the tube is often associated with infection of the gallbladder. Moersch and Camp[41] (1934) stated that intra-abdominal infections, such as cholecytitis, duodenal ulcer and appendicitis, may be carried by lymphatic vessels to the esophagus and may give rise to localized esophageal infection.

39. Jackson, Chevalier: Peptic Ulcer of the Esophagus, J. A. M. A. **92**:369 (Feb. 2) 1929.

40. Mosher, H. P.: Hemorrhage into the Esophagus at Birth and in the Adult, Laryngoscope **41**:591 (Sept.) 1931.

41. Moersch, H. J., and Camp, J. D.: Diffuse Spasm of the Lower Part of the Esophagus, Ann. Otol., Rhin. & Laryng. **43**:1165 (Dec.) 1934.

No work, so far as we have found, has been carried out on the bacteriology of the esophagus and the relation of the organisms found to the type of ulcer produced. Rosenow [42] (1923) was able to produce ulcers in the stomach and duodenum of rabbits by injecting streptococci isolated from infected teeth and tonsils of patients suffering with gastric and duodenal ulcers. No work of this nature has been done with esophageal ulcerations.

In our series of 213 patients, 128 (60 per cent) had had some type of infection (such as peritonitis, pneumonia or cholecystic disease).

Septicemia was present in 16 cases (8.4 per cent). The type of organism found in cultures of the heart blood was as follows: Streptococcus haemolyticus in 7 cases, Staphylococcus aureus in 4, Streptococcus viridans in 3, Proteus ammoniae in 1 and Bacteroides funduliformis in 1. This condition offered a chance for direct infection by way of the blood stream. Periesophagitis was not present in any of the cases of septicemia.

Fifty of the patients (23.4 per cent) had chronic cholecystic disease, the average age of these patients being 54 years. A diagnosis of acute cholecystitis was made in only 2 cases. Peritonitis was present in 76 cases (35.6 per cent) and was general in 59, beginning in 7, pelvic in 5, local in 4 and resolving in 1.

There were 109 cases (51.1 per cent) of pneumonia. These included instances of beginning, early, lobar, terminal, embolic and aspiration pneumonia.

Eleven cases (5.1 per cent) of chronic ulcerative colitis were noted.

According to most workers, cholecystitis, peritonitis, chronic ulcerative colitis and pneumonia may infect the esophagus through the lymphatic structures. Microscopically, in esophagitis dilated lymphatic vessels are often seen filled with bacteria and leukocytes. It is impossible to say, however, whether the infection is being carried to the esophagus or away from the ulcerative lesion already present. Nevertheless, it seems reasonable to suppose that the esophagus as well as other organs of the body can become infected by lymphatic transmission. No doubt the original lesion, which later becomes esophagitis, is often initiated by infection from various sources. The incidence of these infections over the period of six years and four months did not parallel the increased incidence of esophagitis.

Involvement of the Nervous System.—In this series the central nervous system was involved in 24 cases, as follows: by a tumor of the brain in 11, by meningitis in 6, by edema of the brain in 2, by

42. Rosenow, E. C.: The Specificity of the Streptococcus of Gastroduodenal Ulcer and Certain Factors Determining Its Localization, J. Infect. Dis. **33**:248, 1923.

internal hydrocephalus in 2, by multiple sclerosis of the brain and spinal cord in 1, by multiple infarcts of the brain in 1 and by rupture of an aneurysm in 1.

In 17 (70.8 per cent) of these 24 cases there was involvement of the stomach, either petechial hemorrhages or fresh ulcerations, usually in the fundus.

The possible bearing of nerve stimulation on the etiology of esophagitis has been discussed. Only 12.5 per cent of these patients had vomited or had had the gastric contents removed or both. This figure is much lower than that observed in other conditions which have been considered. The involvement of the stomach in 70.8 per cent of these cases tends to confirm the experimental work of other investigators.

If stimulation of the central nervous system is capable of raising the acidity of the gastric content and if at the same time it increases esophageal movement, then its importance as an etiologic factor in esophagitis must not be underestimated.

Other Conditions.—1. Relationship Between Burns and Esophageal Ulcers: This relationship is mentioned frequently in the literature as a possible etiologic factor in the production of esophageal ulcers. Three patients with severe superficial burns in the series studied had acute ulcerative esophagitis. Multiple ulcers and erosions were also noted at and below the cardia and pylorus. One patient had vomited blood a few days before death, and in one instance the ulcer had penetrated through the mucosa of the esophagus. In the other case a small amount of blood was present in the stomach which appeared to have come from bleeding ulcers in the esophagus.

It has been known for some time that ulcers of the duodenum are often associated with severe superficial burns. According to Huwald [43] (1893), Fischer [44] early pointed out the similarity between duodenal and esophageal ulcerations, namely, the occurrence of ulcerations of the esophagus following external burns. Huwald expressed the opinion that the lesions are probably caused by the disintegration of the erythrocytes which results in thrombosis or embolism. It is not within the scope of this paper to review all the literature relating to the possible etiology of ulcers associated with external burns. Some of the more recent work, however, will be considered. Novak [45] in 1925 presented a case of gastro-intestinal ulceration following cutaneous burns. He gave a complete report of the postmortem examination except that he

43. Huwald, K., quoted by Tileston.[31]

44. Fischer, quoted by Tileston.[31]

45. Novak, Edward: Gastro-Intestinal Ulceration Following Cutaneous Burns, with Report of Case, Am. J. M. Sc. **169**:119 (Jan.) 1925.

did not mention the esophagus. Pack,[46] in his excellent review of the subject in 1926, under the heading gastro-intestinal tract, stated: "There is a generalized hyperemia of the mucous membrane, with punctate areas of petechial hemorrhage into the mucous membrane of the stomach and intestines, particularly in the ileum near the cecum." Again the esophagus was not mentioned. In 1881 Boyd[47] noted the rupture of the esophagus in a case of Addison's disease, but not until recently has this relation between gastro-intestinal ulceration and injury to the adrenal glands been investigated. Greenwald and Eliasberg[48] (1926) noted that burns produce injury to the adrenal glands. In the early stages there is hyperglycemia as a result of stimulation of the adrenal glands. In the second stage there is hypoglycemia, presumably because of degenerative changes in the adrenal glands. McLaughlin[49] in 1933 produced ulcers in the small bowel of dogs after cauterization of the adrenal glands. No ulcers, however, were seen proximal to the pyloric ring. Later, in 1935,[50] he showed that, in contradistinction to Cushing's[51] theory that vagal stimulation is a factor, he could section the vagus nerves bilaterally and still produce ulcerations by injury of the adrenal glands.

2. Peptic Ulcer: The presence of peptic ulcers with esophagitis has been noted by a number of observers. In this series there were 42 patients with peptic ulcer (20.1 per cent); 36 were males and 6 were females. Twenty-one (50 per cent) had vomited or had had the gastric contents removed by tube or both. There were only 4 (16 per cent) gastric ulcers, the remainder being duodenal. The exact reason for this relationship is not known. Possibly, in both these conditions the acids of the gastric content play an important rôle. This no doubt is true only when for some reason there is an insufficient protection of the mucous membrane by the secreted mucus.

3. Arteriosclerosis: Mosher[52] (1934) expressed the opinion that fibrosis of the esophagus is common in such degenerative diseases as

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46. Pack, G. T.: The Pathology of Burns, Arch. Path. & Lab. Med. **1**:767 (May) 1926.

47. Boyd, Stanley: Rupture of the Oesophagus in a Case of Addison's Disease, Tr. Path. Soc. London **33**:125, 1882.

48. Greenwald, H. M., and Eliasberg, H.: The Pathogenesis of Death from Burns, Am. J. M. Sc. **171**:682 (May) 1926.

49. McLaughlin, C. W.: The Curling Ulcer: Study of Intestinal Ulceration Associated with Suprarenal Damage, Arch. Surg. **27**:490 (Sept.) 1933.

50. McLaughlin, C. W.: Intestinal Ulceration Following Adrenal Damage: Experimental Observations, Ann. Surg. **101**:554 (Jan.) 1935.

51. Cushing, Harvey: Peptic Ulcers and the Interbrain, Surg., Gynec. & Obst. **55**:1 (July) 1932.

52. Mosher, H. P.: Notes on Esophagus Cases, Ann. Otol., Rhin. & Laryng. **43**:1154 (Dec.) 1934.

arteriosclerosis. Miller [53] (1912) said that a poor blood supply, together with the action of the acid gastric juice, accounts for the ulcerations seen in the thoracic section of the esophagus. It is well known that a reduced blood supply results in an increased hydrogen ion concentration, which greatly facilitates autolysis.

In this series of cases the degree of arteriosclerosis, on a basis of 4, averaged 1 plus. Over the period of six years and four months there was no increase in the degree of arteriosclerosis. Slowing of the blood stream may lead to thrombosis and infarction; thus an area of devitalized tissue becomes a target for infection or irritative gastric contents.

4. Abnormal Alteration in the Constituents of the Blood: Vinson and Wilder [54] in 1933 reported cases of diffuse ulceration of the esophagus and trachea associated with diabetes mellitus. In our series 9 patients had a blood sugar content greater than 120 mg. per hundred cubic centimeters, the average being 307 mg. Four (44.4 per cent) of these patients had vomited or had had the gastric content removed or both. In all 9 cases a diffuse ulceration was present in the lower third or lower half of the esophagus. In no case was ulceration of the trachea observed.

The possible etiologic relationship of urea also was considered. There were 48 cases in which the blood urea content was more than 50 mg. per hundred cubic centimeters, the average being 162.4 mg. Twenty-four (50 per cent) of these patients had vomited or had had the gastric contents removed or both.

Twelve patients had a serum bilirubin value greater than 3 mg. per hundred cubic centimeters, the average being 16.6 mg. Five (41.6 per cent) of these patients had vomited or had had the gastric contents removed or both.

It appears that, although these excess chemical factors in the blood may play a part by reducing the resistance of the tissue or allowing hemorrhage, the high percentage (45.3 per cent) of these patients who had vomited or had had the gastric contents removed or both leads one to feel that this is of equal importance.

5. The Rôle of Operation: In 159 (74.6 per cent) of the cases in this series esophagitis followed operation. Just what direct effect an operative procedure has on the production of esophagitis is not known. Moutier [55] in 1921 reported 3 cases in which symptoms of esophagitis

53. Miller, James: A Case of Perforated Peptic Ulcer of the Oesophagus Complicated by Pyopneumothorax, Brit. M. J. **1**:116 (Jan. 20) 1912.

54. Vinson, P. P., and Wilder, R. M.: Diffuse Ulceration of the Esophagus and Trachea Associated with Diabetes Mellitus: Absence of Arteriosclerosis, Arch. Int. Med. **52**:541 (Oct.) 1933.

55. Moutier, F.: Acute Postoperative Esophagitis, Arch. d. mal. de l'app. digestif **11**:126, 1921; abstr., J. A. M. A. **76**:1536 (May 28) 1921.

developed following operation. These symptoms lasted for four days and then subsided. Probably the relaxation produced by anesthesia allows for a backflow and stasis of the gastric contents which initiate the process. The curve plotted for these cases of postoperative esophagitis over a period of six years and four months did not parallel the increased incidence of esophagitis.

Symptoms.—1. Esophagodynia: The most common symptom of esophagitis is described by the patient as a "burning in the chest." This burning pain is most often in the back and lower third of the sternum. Eusterman, Moersch and Camp [56] reported a case of esophageal ulceration in 1930 in which there was a history of both gastric and duodenal ulcer. Rivers [57] (1935) found that 45 per cent of his patients with esophageal ulcer gave a history of peptic ulcer.

The type of pain experienced and the method of its transmission are still undetermined. Kahn [58] (1906) noted that the irritability of the esophagus increased from the pharynx to the cardia. Hurst [59] (1911 and 1929) found that the esophagus was sensitive to heat and cold but insensitive to tactile sensations. He further observed that dilute hydrochloric acid or dilute organic acids cause no sensation in the lower third of the esophagus, regardless of whether or not the mucous membrane is intact. Alvarez [37] (1928), however, suggested that the pharyngeal end of the esophagus is sensitive and supported this view with the well known fact that following vomiting there is the so-called water brash.

Boring [60] (1915) also observed that the mucous membrane of the esophagus is not very sensitive to irritants, but he could not explain the painful sensations that are sometimes felt, which are usually referred upward into the neck or downward into the epigastrium. Polland and Bloomfield [61] (1931) found experimentally that the inflation of small balloons in the esophagus produced sensations of referred pain. The site of the referred pain had no relation to the position of the balloon. Most often the pain occurred at the xiphoid process or in the supra-

56. Eusterman, G. B.; Moersch, H. J., and Camp, J. D.: Peptic Ulcer at the Juncture of the Esophagus and Cardia: Ulceration at the Esophagus, M. Clin. North America **14**:565 (Nov.) 1930.

57. Rivers, A. B.: Pain in Benign Ulcers of the Esophagus, Stomach, and Small Intestine, J. A. M. A. **104**:169 (Jan. 19) 1935.

58. Kahn, R. H., quoted by Alvarez.[37]

59. Hurst, A. F.: The Sensibility of the Alimentary Canal, London, Oxford University Press, 1911, pp. 4, 9, 15 and 35. Hurst, A. F., and Stewart, M. J.: Gastric and Duodenal Ulcer, London, Oxford University Press, 1929, pp. 43 and 498.

60. Boring, E. G., quoted by Alvarez.[37]

61. Polland, W. S., and Bloomfield, A. L.: Experimental Referred Pain from the Gastro-Intestinal Tract: I. The Esophagus, J. Clin. Investigation **10**:435 (Aug.) 1931.

sternal notch, and less often, over the anterior wall of the chest or in the back. Rarely was it felt in the neck or face. In several cases the authors were able to produce nausea. They expressed the opinion that their results were not accurate, because of the variations which occurred almost from moment to moment. These, they considered, depended on psychic factors and on the ever changing conditions of tonus and motility which are encountered in a hollow viscus.

Payne and Poulton [62] (1927) concluded that visceral pain in the esophagus results from a stretching and deformity of the nerve endings in the wall. They observed that the stretching force that causes this pain is relieved by a peristaltic contraction. The pain can be made worse by a contraction which does not overcome the stretch. They said that the "burning pain" which is sometimes experienced results from a continuous stretching and deformity of the nerve endings.

Moersch and Camp [41] in 1934 reported 8 cases of diffuse spasm of the lower part of the esophagus. Pain and dysphagia were the outstanding symptoms. In only 1 case was a small superficial ulcer demonstrated by esophagoscopic examination. Application of silver nitrate to that area produced pain which was referred to the right shoulder.

In our series substernal burning was present in 17 cases (53.2 per cent).

2. Hematemesis: Hematemesis and melena have been reported as symptoms occurring in the course of esophagitis. In a new-born child Henoch [63] (1883) observed melena associated with lesions in the lower part of the esophagus, from which the bleeding originated. Bastin [64] (1908) also gave esophageal ulcers as the cause of this type of melena.

Bristowe [65] (1856-1857) described a case of hemorrhage from a varicose ulcer of the esophagus which produced death. Cabot [66] in 1931 and many other investigators have reported numerous similar cases.

Hematemesis is probably the most common of the three types of bleeding mentioned. Pringle and Teacher [67] (1919) have emphasized

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62. Payne, W. W., and Poulton, E. P.: Experiments on Visceral Sensation: II. The Sensation of "Nausea" and "Sinking," Oesophageal Reflexes and Counter-Irritation, J. Physiol. **65**:157 (May) 1928.

63. Henoch, E.: Verhandlungen ärztlicher Gesellschaften, Berl. klin. Wchnschr. **20**:334 (May 28) 1883.

64. Bastin, F. K.: Ueber die Verstopfung des Oesophagus durch ein Blutgerinnsel bei Malaena neonatorum, Munich, Kastner & Callwey, 1908, p. 44.

65. Bristowe: Varicose Ulcer of the Oesophagus, Producing Death by Haemorrhage, Tr. Path. Soc. London **8**:175, 1856-1857.

66. Cabot, R. C.: Massive Hematemesis and Anemia in a Woman of Fifty-Five, New England J. Med. **204**:1000 (May 7) 1931.

67. Pringle, J. H., and Teacher, J. H.: Digestion of the Oesophagus as a Cause of Postoperative Haematemesis, Brit. J. Surg. **6**:523, 1919.

the importance and danger of postoperative hematemesis. The symptoms usually become manifest soon after operation. Some observers have suggested that this bleeding is due to sepsis and alteration in the blood. Thus, the blood is capable of exuding through an apparently intact mucous membrane. Anesthetic agents have been blamed by some writers. No doubt they do often excite vomiting, but how they could produce bleeding we cannot understand.

In our series hematemesis occurred in 15 cases (46.8 per cent). It was the second most frequent symptom. The amount of blood vomited varied from a few cubic centimeters to 200 cc. A few of the patients vomited blood on several occasions. Grossly all the lesions were acute and ulcerative and involved the lower third of the esophagus. In 1 case the entire esophagus was involved. In 1 patient who had vomited blood a ruptured varix was found. In none of the cases of hematemesis was the esophagus suspected clinically as being the source of the bleeding. At necropsy no other lesion in the upper part of the alimentary tract was observed which could account for the bleeding except the ulcerated esophagus.

3. Dysphagia: Dysphagia is usually accompanied with pain. The spasm resulting from the presence of the inflammation is the cause of this symptom. The physiology and pathology of this condition have been discussed in detail in this paper.

In our series dysphagia was present in 9 cases (28.1 per cent). It occurred frequently as an accompaniment of substernal pain. In a few cases it was so severe that tube feeding was necessary.

4. Analysis of Symptoms Observed in This Series: In our series of cases of esophagitis there were 136 males and 77 females. The oldest patient was 82 years old, the youngest 2 months. There were definite clinical symptoms in 32 cases (10.3 per cent) which suggested esophagitis. Twenty-four of these patients (72 per cent) had vomited or had had the gastric contents removed or both. In 23 cases (71.9 per cent) esophagitis followed operation, the symptoms usually appearing from twenty-four to forty-eight hours afterward and continuing for from three to ten days. In the cases in which operation was not performed the symptoms usually began several days before death. In a few of the cases in which the gastric contents were removed the symptoms appeared a short time later. In 1 case the diagnosis of acute ulcerative esophagitis was made clinically on the history of severe substernal burning.

Clinically the esophagus was not considered, except in the 1 case described, as being the cause of the substernal pain or dysphagia. All the information obtained was volunteered by the patient to the doctors

in charge. It seems reasonable to suppose that if patients had been questioned as a routine concerning these symptoms the percentages would be much higher.

Diagnosis.—Substernal burning and pain, hematemesis and dysphagia are the most common symptoms. Whenever any of these symptoms occur the diagnosis of esophagitis should be considered.

Obstruction is sometimes a late symptom of esophagitis. The spasm produced by esophagitis may lead to hypertrophy of the muscular coats, decompensation and, later, dilatation of the tube. As Barclay [68] pointed out in 1933, the patient with esophageal ulcer may go through all the stages of hypertrophy, pain, obstruction and starvation, and he often remains comfortable in a state of semistarvation. The contraction of the esophagus caused by spasm from the ulcer often makes eating difficult. This seems to explain why carcinoma is so often suspected.

Jackson, in discussing Winkelstein's [69] report in 1935, stated that esophagoscopy is indicated in every case in which unyielding gastric symptoms are present. Levine [70] (1929) also found this method of use in the diagnosis in his cases.

Barclay [25] and others have pointed out the importance of roentgenography as a diagnostic procedure. Usually there is seen spasm of the lower third of the esophagus without dilatation. Belladonna, according to Barclay, relieves the spasm in some cases, whereas in others it has no effect. The severity of the spasm appears to depend not on the size of the ulcer but on its irritability. Otell and Coe [71] in 1935 described spasm as the chief roentgenographic characteristic of ulcer of the esophagus: "The roentgen appearance in acute esophagitis shows no variation from the normal."

In summary, esophagitis should be suspected when any of the foregoing symptoms appear. Hematemesis at any time, but especially after an operation or an acute illness, should make one suspect that the esophagus is its source. It is probably a good rule to perform esophagoscopic examination whenever there are unyielding gastric symptoms. The importance of distinguishing between esophageal cancer and obstruction due to esophagitis cannot be overemphasized.

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68. Barclay, A. E.: The Digestive Tract: A Radiological Study of Its Anatomy, Rhysiology, and Pathology, London, Cambridge University Press, 1933, p. 177.

69. Winkelstein, Ashur: Peptic Esophagitis: A New Clinical Entity, J. A. M. A. **104**:906 (March 16) 1935.

70. Levine, S. A.: A Case of Ulcer of the Esophagus with Extraordinary Medical Experiences, M. Clin. North America **13**:189 (July) 1929.

71. Otell, L. S., and Coe, F. O.: Dysphagia—Roentgenologically Considered, Am. J. Digest. Dis. & Nutrition **2**:117 (April) 1935.

COMMENT AND SUMMARY

It has been shown that because of its anatomic structure the esophagus is vulnerable to the organisms of the oral cavity, the regurgitated gastric contents and infection through lymphatic structures from the abdominal viscera.

Experimentally it has been proved that the mucus which normally covers the lining of the tube is the greatest protective barrier. Lesions heal quickly in a normal esophagus, but without the protective mucous layer and with slight irritation the lesions go on to form ulcers. Microscopically one often observes that the mucous glands are blocked and dilated or have been destroyed by infection, hemorrhage or thrombosis. In this manner the supply of mucus is diminished. Rapid dilatation and strong movement of the tube, as a result of vomiting or stimulation of the central nervous system, removal of gastric contents by tube or infarction are all capable of producing esophageal lesions. These lesions, with poor protection owing to diminished secretion of mucus, are constantly being irritated by normally regurgitated gastric content. Thus the inflammatory process is initiated.

In our series vomiting and the removal of gastric contents were the only factors which showed a relation to the increased incidence of esophagitis. However, infection, stimulation of the central nervous system and a poor blood supply are all factors which may initiate a lesion.

Postmortem gastric analyses show that during the last few hours before death there are not sufficient irritative properties in the gastric content to produce esophagitis seen at the autopsy table. Esophagitis, then, is not entirely an agonal condition.

However, vomiting, we believe, is the important factor in the etiology of esophagitis. It seems reasonable to suppose that recurrent attacks of esophagitis occur more frequently at sites where healing, with the production of fibrous tissue, has occurred. Since normal persons are constantly regurgitating gastric contents into the esophagus, it seems reasonable to suppose that during a lifetime there may occur many unrecognized attacks of esophagitis. When healing occurs fibrous connective tissue forms. If this process occurs frequently enough a benign stricture may result.

In the 3,032 necropsies performed, the incidence of esophagitis was 7.02 per cent. In 75.2 per cent of the cases the lesions were found in the lower third of the esophagus. In 76.5 per cent of the 213 cases in which it was present the esophagitis was of the acute ulcerative type. Definite symptoms occurred in 10.3 per cent of the cases, and the most common symptom was substernal burning, which occurred in 53.2 per cent.

OPERATIVE EXPOSURE OF THE FACIAL CANAL

WITH REMOVAL OF A TUMOR OF THE GREATER SUPERFICIAL PETROSAL NERVE

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A case of operative exposure of the facial canal with removal of a tumor of the greater superficial petrosal nerve is reported in order to describe the procedure and because no other description of a perineurial fibroblastoma arising in the vicinity of the geniculate ganglion is to be found in the literature.

The presenting symptom was facial paralysis. But preliminary tests localized the site of interruption to the genu of the facial nerve, and exploration was undertaken with a view to a possible end-to-end suture within the petrous bone.

In 1908 Alt [1] suggested a means of opening the facial canal, and in 1922 Ney [2] reported a method of decompression of the nerve and suture worked out on the cadaver. The more recent work of Ballance and Duel [3] on nerve grafts in the treatment of facial palsy contains also a complete bibliography of the subject.

REPORT OF CASE

William N., aged 42, was admitted to the Montreal Neurological Institute on Jan. 28, 1935, complaining of left facial paralysis of five years' duration and of tinnitus and partial deafness of the left ear of two and a half years' standing.

In the latter part of 1929 while working for the government as a construction engineer in Texas, the patient had his attention called to the fact that the left side of his face was partially paralyzed. During the next year the paralysis gradually progressed until it was complete. He then moved to New York and there

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From the Departments of Otolaryngology and of Neurology and Neurosurgery of McGill University and the Montreal Neurological Institute.

1. Alt, F.: Verhandl. d. deutsch. otol. Gesellsch. **17**:191, 1908.

2. Ney, K. W.: Facial Paralysis and the Surgical Repair of the Facial Nerve, Laryngoscope **32**:327, 1922.

3. Ballance, C., and Duel, A. B.: The Operative Treatment of Facial Palsy by Introduction of Nerve Grafts into the Fallopian Canal and by Other Intratemporal Methods, Arch. Otolaryng. **15**:1 (Jan.) 1932.

had electrical treatment at the New York Neurological Institute. The paralysis gradually improved over the period of a year but at the end of that time complete paralysis returned.

Two years after the first appearance of the facial weakness, he began to hear a constant buzzing in his left ear, "just like the buzzing of a bee." This was present without intermission up to the time of his admission to the Montreal Neurological Institute. With the onset of this tinnitus, he noticed that his hearing gradually became less acute until the time of his admission, when he could just hear a watch held close to his ear. He had not noted otorrhea or otalgia. He had never complained of dizziness, nystagmus, vertigo or difficulty in walking.

Examinations.—In the routine examination of the nose and throat, it was noted that far back on the nasal septum there was a spur; the anterior cartilaginous part of the septum had been removed. No evidence of sinusitis was found. The tonsils had been well removed; the findings in the pharynx were otherwise negative. The nasopharynx was clear. The hypopharynx and the larynx were normal. The vocal cords were normal and moved equally well. The results of the general physical examination were negative. The reflexes were normal. The cranial nerves were normal except as described in the following paragraphs:

Fig. 1.—Patient with tumor of the greater superficial petrosal nerve.

Facial Nerve: There was paralysis of the left side of the face, including the upper as well as the lower portion. There was also slight contraction in the frontalis muscle on the left side (fig. 1). There was also occasional involuntary movement of the left angle of the mouth, indicating an attempted regeneration of the nerve.

The results of the examination of the chorda tympani were as follows:[4] Concentrated salt solution on the right side of the tongue was identified in five seconds; the same solution on the left side of the tongue was not identified after

4. Dr. T. C. Erickson, the neurosurgical resident, is responsible for the ingenious examinations of function in the chorda tympani and greater superficial nerves.

one minute. Vinegar was suggestively better recognized on the right than on the left, but the results varied. Sugar was not satisfactorily tasted on either side. The posterior third of the tongue showed no difference in recognition of salt, vinegar or sugar on the right and left sides. The conclusion was that taste was impaired over the anterior portion of the tongue on the left.

On direct observation of the orifices of the excretory ducts of the parotid glands, that on the right was found to be elevated on a papilla much larger than that on the left, and the gland on the right appeared to secrete more actively than the one on the left. Quantitative measurement of the secretion for three minutes after the stimulation with vinegar showed equal secretion on the two sides, i. e., 0.11 Gm. on the right and 0.11 Gm. on the left. The estimation of secretion from the submaxillary and sublingual glands was as follows: right, 0.61 Gm.; left, 0.17 Gm. Thus there was shown to be a very marked diminution in secretion from the left submaxillary and sublingual glands, due apparently to an interruption of fibers of the chorda tympani to these glands in their passage within the seventh nerve.

The conclusion was that there was impairment of fibers of the chorda tympani on the left side, shown by the loss of taste and the decrease of salivary secretion, but not of the fibers supplying taste to the posterior third of the tongue or of the fibers supplying the parotid gland (both of which are innervated by the ninth cranial nerve).

The greater superficial petrosal nerve was examined as follows: A quantitative estimation of the lacrimal secretion was made by means of a tampon placed near the external canthus of each eye without local use of an anesthetic. The weight of the tampon before and after was determined. The secretion of tears was stimulated by means of ammonia and the difference from the normal amount measured. The right gland secreted 0.055 Gm. for a period of two minutes, and the left gland, 0.001 Gm. This showed a marked diminution of lacrimal secretion on the left side as compared with the right (1 : 50) and was evidence of an involvement of the facial nerve at or proximal to the geniculate ganglion or of an involvement of the greater superficial petrosal nerve.

Auditory Nerves: A watch was heard at 12 inches (30.5 cm.) on the right and could not be heard on the left side. Air conduction was greater than bone conduction, but in Weber's test the patient insisted that the sound was lateralized to the left. He said that he did not confuse the sound with the tinnitus which was ordinarily present.

The right drum showed the auditory meatus to be clear. The drum was pearly gray, and all the landmarks were present. The left auditory meatus showed three exostoses, two of them on the anterior wall, which partially obstructed the view of the drum. Apart from two chalky deposits superiorly, the tympanic membrane was normal with a clear light reflex.

The audiogram showed a loss of only 10 sensation units in each ear for conversational tones (i. e., tones of from 64 to 512 double vibrations) before and after catheterization. However, for the high notes (8,192 double vibrations) the hearing in both ears was affected. There was a loss of 35 sensation units in the left ear.

The caloric test showed a normal reaction with 2.5 cc. of cold water on the right side. On the left side 30 cc. of cold water gave only a sluggish reaction. These findings were corroborated by means of the Bárány chair, in which ten turns to the right gave a moderate nystagmus to the left with only slight dizziness. The same number of turns to the left gave an active nystagmus to the right with marked dizziness.

In summary, therefore, the patient had partial paralysis of the left auditory nerve with air conduction greater than bone conduction and the sound in the Weber test referred to the left. Vestibular responses were sluggish on the left.

The left facial nerve was completely paralyzed except for a few fibers coming through to the left angle of the mouth. These were regenerated fibers, as indicated by the fact that the angle of the mouth moved only involuntarily. The chorda tympani was paralyzed, as indicated by the decreased secretion of saliva from the sublingual and submaxillary glands on the left and the loss of taste in the anterior two thirds of the tongue. The greater superficial petrosal nerve, which leaves the facial nerve at the geniculate ganglion, was also paralyzed, as

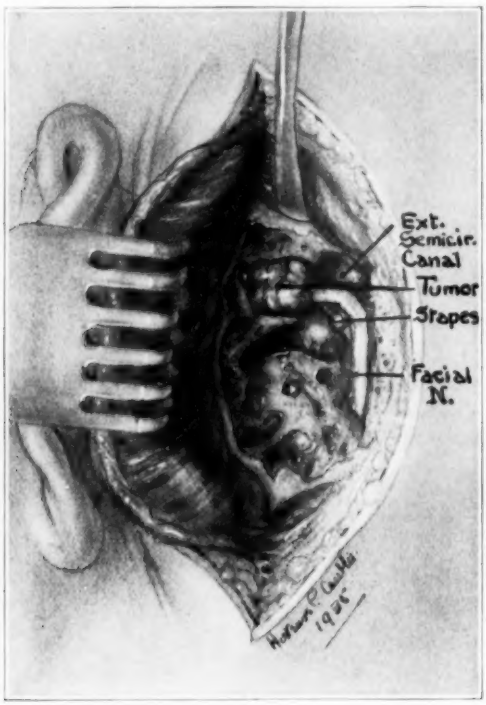

Fig. 2.—Field of operation.

indicated by the lack of increased secretion of tears on stimulation of the left conjunctiva. The site of interruption of the seventh nerve was thus obviously as far proximal as the geniculate ganglion.

Operation.—It was decided to expose the left facial nerve in its vertical and horizontal portions and to incise the sheath. If no organic cause was found for the paralysis, an anastomosis of the peripheral segment of the facial nerve with the hypoglossal nerve was to be contemplated.

On Feb. 12, 1935, the operation was carried out under tribromethanol in amylene hydrate and a locally applied anesthetic. The same incision was made as for a radical mastoid operation except that it was extended a little above and below. All the mastoid cells were exenterated widely, and the whole tip of the mastoid process was removed as well as the vaginal process of the external

auditory canal. The cells over the lateral sinus were uncovered, but the sinus itself was not exposed. The middle ear was opened from behind by enlarging the aditus ad antrum and removing part of the roof of the external meatus. After identifying the horizontal semicircular canal all the bone below this point as far as the stylomastoid foramen was taken away. This is not a difficult or dangerous procedure, as the vertical portion of the facial canal lies in the same plane as the horizontal semicircular canal. The whole canal was uncovered, exposing the facial nerve. In order to get a better exposure of the nerve, some of the bony floor of the middle fossa was removed. There was some troublesome bleeding in this

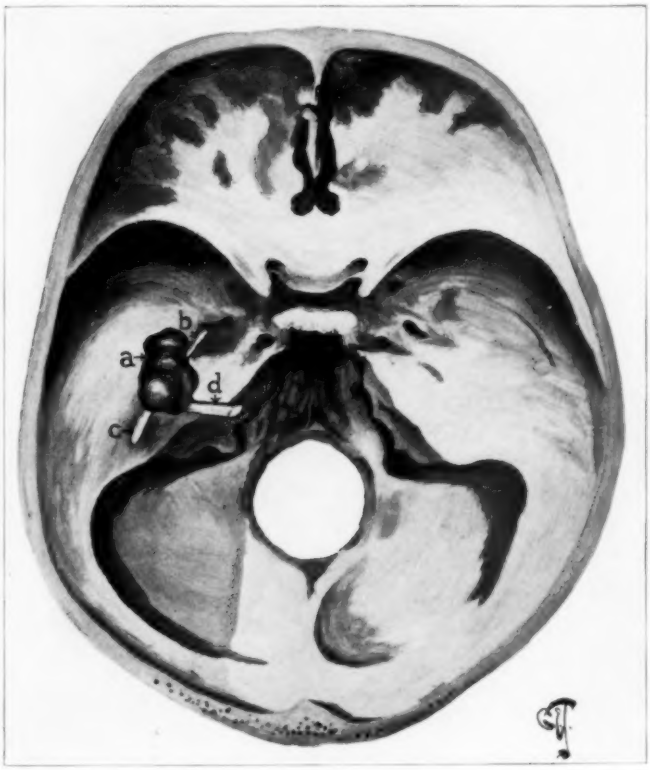

Fig. 3.—Drawing showing (*a*) tumor involving (*b*) greater superficial petrosal nerve, (*c*) facial nerve and (*d*) seventh and eighth nerves.

situation, evidently from the posterior branch of the middle meningeal artery, but it was easily controlled. The facial nerve was identified again at the bend and traced along its horizontal course. The stapes immediately below the nerve was found but carefully avoided.

At a point about 4 mm. from the bend and just above the oval window, the nerve was involved by a vascular mass of tissue, which proved to be a new growth. On removal of the roof of the middle ear, a tumor herniated into the opening. It had extended through the bone from the facial canal to the subdural space, apparently following the canal of the greater superficial petrosal nerve.

The main portion of the tumor thus presented beneath the dura on the anterior aspect of the petrous pyramid. As much of the growth as possible was removed, and part of this was sectioned at once. During this procedure, the incus was gently taken away with the curet, and, later in the operation, the malleus and the drum. The tumor was curetted out and the seventh nerve left exposed without incision of the nerve capsule. Finally the eustachian tube was curetted so that it would close off with a view to future complete extirpation of the tumor. At no time was there any escape of cerebrospinal fluid. The wound was closed with two drains and the external auditory meatus left open as in a radical mastoid operation.

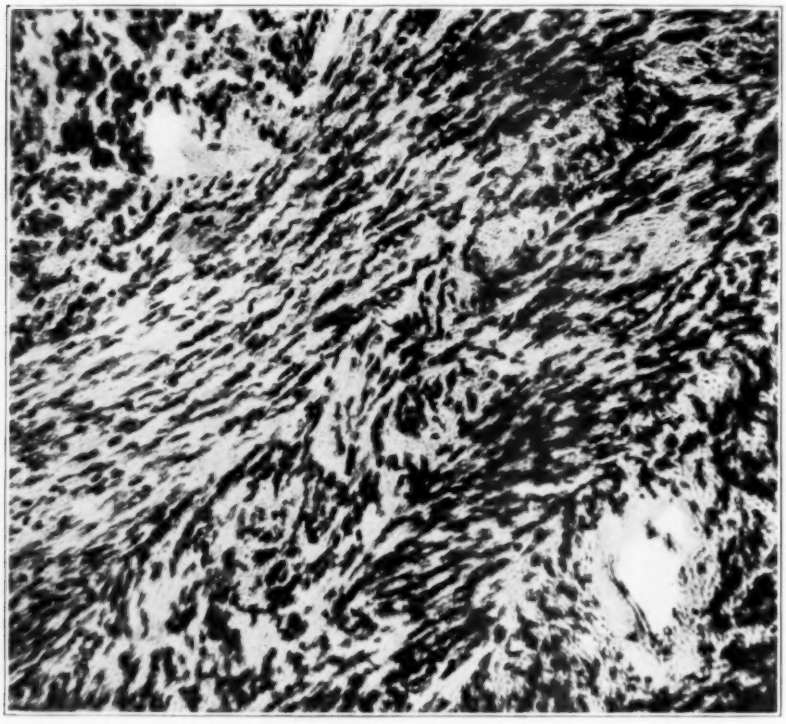

Fig. 4.—Cross-section of tumor.

It was decided not to make an anastomosis of the seventh nerve to the twelfth as it seemed quite possible that the removal of the tumor would allow the seventh nerve to regenerate. It is hoped that if it becomes necessary to reoperate on this tumor at some future time the closure of the eustachian tube will be sufficient to allow the whole procedure to be carried out without fear of contamination. Complete removal would have involved removal of the dura in the presence of a contaminated field.

Pathologic Report.—The tumor was made up of small fragments not over 5 mm. in diameter. Some of these were yellowish white and others light red. All were soft. There was no evidence of calcification.

Microscopic examination showed the tumor cells to have moderately abundant cytoplasm, spindle-shaped in outline and staining deeply with hematoxylin and

Van Gieson's stain. The nuclei were small and round or elongated, rich in chromatin, lacking a nucleolus and uniform in size. The cells were loosely arranged for the most part, but there were a few very dense streams. No mitotic figures were seen. When the cells occurred in streams, bipolar processes were found.

Blood vessels were numerous, occurring usually in small clusters, and practically all had heavy deposits of hyalin in their walls. Fibrous connective tissue was abundant, and there were many areas of almost pure collagen. Other broad bands streamed through the section. There was at least one large area of old hemorrhage but no necrosis, cystic formation or calcification. There were large areas of fibrosis. Laidlaw's connective tissue preparation demonstrated many streams of parallel wavy fine fibers, characteristic of perineurial fibroblastomas.

The diagnosis was perineurial fibroblastoma.

COMMENT

The clinical tests described in this paper should be found of help in localizing the site of an injury of the facial nerve regardless of what the cause of that injury may be.

The otogenic causes of paralysis may be acute or chronic inflammation of the middle ear, labyrinthitis, malignant growths or operative injury. Paralysis is often due to a chronic suppurative otitis media in which the bony sheath becomes eroded by a cholesteatoma. Most frequently operative injury is done when removing the bridge in the radical mastoid operation. Injury may also be produced by using packing which is too tight, by curetting the middle ear in the vicinity of the nerve or, in infants, by extending the initial incision of the skin down too low. In all these conditions, exact localization of the site of injury would throw light on the therapy.

It is believed that no other case of perineurial fibroblastoma (neurinoma) of the greater superficial petrosal nerve has been recorded in the literature, although there is no reason why such a tumor should not grow in this situation. Curiously enough, a tumor of this type, although common on the vestibular portion of the eighth nerve, is very rare on any other cranial nerve. The fifth cranial nerve is the next most frequent site. The tumor in the case presented was discovered only in the course of the exploration of the facial canal.

The technic of the exploration is described, and it is urged that such an exploration should be carried out in many cases of paralysis of the facial nerve with a view to (1) relief of the pressure on the facial nerve, (2) direct suture of the nerve or (3) facial-hypoglossal anastomosis, as indicated by the operative findings.

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PAGET'S DISEASE AND DEAFNESS

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The study of Paget's disease of bone from an otologic point of view is important because of the impairment of hearing which occurs in some instances of this disease and more especially because of the similarity of the histopathologic picture to that of otosclerosis.

Osteitis deformans, or Paget's disease, was first described by Sir James Paget in 1877. By 1906 about seventy cases had been recognized. Since then a large number of cases of this disease have been studied both clinically and pathologically, particularly since the widespread use of roentgen examination in diagnosis.

In some of the patients with early phases of the disease deafness was noted, but the interest of otologists was not aroused until Otto Mayer [1] presented his studies pointing out the similarity of the histologic picture of Paget's disease with that of otosclerosis. In 1913 he published a report of a clinical case with results of hearing tests; the following year he presented a pathologic study of a temporal bone with Paget's disease. By 1917 he was able to report on a study of nine cases of the disease in which there was impairment of hearing; the reports of two of these cases included the results of histopathologic studies of the temporal bone. Nager [2] made a similar contribution in 1919. In 1923 Jenkins [3] presented a clinical report of nine cases, including histopathologic studies of two cases. The same author reported observations on a larger group of cases in 1928. Further studies have since been reported by Nager and Max Meyer,[4] Brunner [5] and Weber.[6]

From the Division of Otolaryngology, Department of Surgery, the University of Chicago.

1. Mayer, Otto: Internat. Zentralbl. f. Ohrenh. **11**:530, 1913; Untersuchungen über die Otosclerose, Monograph, Vienna, Alfred Hölder, 1917.

2. Nager, F. R.: Ztschr. f. Ohrenh. **78**:195, 1920.

3. Jenkins, G. J.: J. Laryng. & Otol. **38**:344, 1923; **43**:1, 1928.

4. Nager, F. R., and Meyer, Max: Beitr. z. Anat., Physiol., Path. u. Therap. d. Ohres **30**:169, 1931.

5. Brunner: Ztschr. f. Hals-, Nasen- u. Ohrenh. **30**:128, 1931.

6. Weber, M.: Otosclerosis in Its Histogenic Relations to Osteodystrophia Fibrosa (Ostitis Fibrosa), Arch. Otolaryng. **11**:1 (Jan.) 1930.

In 1934 Barth [7] reported four cases and reviewed twenty-four cases from the literature, including cases of both Paget's and Recklinghausen's disease of bone. In six cases there was inner ear deafness, and in eight combined middle ear and inner ear deafness was present. Barth found that vestibular symptoms played even a greater rôle than disturbances of hearing in these cases. During the past few years in the clinic of the department of surgery of the University of Chicago, of twenty-five patients with Paget's disease, seven of whom had involvement of the skull, four presented definite impairment of hearing. A short report of these cases with results of hearing tests is presented.

REPORT OF CASES

Case 1.—Mr. C., aged 59, appeared at the clinic on June 7, 1932, stating that he had suffered from a buzzing sensation in the ear for seven weeks, cramplike pains in the legs for two years and stiffness and tenderness of the thighs for three weeks. Two years before the onset of his present difficulty he had a slight earache—he did not remember on which side—lasting about three days. No further complaints were noted until seven weeks before he came to the clinic, when deafness and buzzing, together with mild pain in the ear, were noticed. A tonsillectomy was performed, but the buzzing became worse, involving the other ear and keeping the patient awake. The buzzing was synchronous with the pulse. No dizziness was noted. There was no family history of deafness. One sister died of diabetes at 60, and one brother died of arteriosclerosis at 70.

The general physical examination revealed a moderate enlargement of the thyroid gland and carious teeth; the heart and lungs were normal. Laboratory tests, including a Wassermann test of the blood, blood count and determination of the calcium and the phosphorus content of the blood gave normal results. There was a trace of albumin in the urine.

Roentgen studies revealed typical lesions of Paget's disease involving the pelvis, skull and femurs. The involvement of the skull as described by the roentgenologist consisted of a "thickening of the bones of the vault along with alternating spherical patches of increased and decreased density." The examination revealed intact ear drums with some scarring and thickening. The canals were normal. The audiogram is shown in figure 1. The response of the labyrinths to the caloric test with cold water was within normal limits.

Case 2.—Mrs. P., aged 50, appeared at the clinic on April 15, 1931, complaining of anterior bowing of the bones of the legs and gradual reduction in height from 5 ft., 9 inches to 5 ft., 5½ inches (175.26 to 166.37 cm.). She had noted an enlargement of her head for the past ten years. She began having aural symptoms fifteen years before, when tinnitus was noted. No impairment of hearing occurred until six years before, but deafness had grown rapidly worse since then. Tinnitus consisted of three distinct types of sounds, but there was no vertigo. There was no history of otitis media. The tonsils were removed eight years before because

7. Barth, H.: Ztschr. f. Hals-, Nasen- u. Ohrenh. **35**:305, 1934.

of tonsillitis. On several occasions the eustachian tubes were inflated without changing the symptoms. There was no history of deafness in the family. A general physical examination revealed nothing of importance except the skeletal changes which were verified by roentgen examination.

The Wassermann reaction of the blood was negative. Urinalysis and determination of the calcium and phosphorus content of the blood gave normal results.

The roentgenologist described the involvement of the skull on April 16, 1931, as follows: "There is evidence of typical Paget's disease involving the entire vault of the skull. The lesions consist of gross thickening of the skull plus a mottling that is produced by rough spherical areas of increased density, ranging in size from a few millimeters to over 0.5 cm., interspersed with ragged areas of decreased density." He reported the presence of lesions in the skull, thighs, legs and pelvis.

Examination revealed the ear drums to be normal, but a diffuse pinkish hue was visible through both drums. The results of the hearing tests are given in figure 2.

CASE 3.—Mr. B., aged 59, was first seen at the clinic on March 18, 1931, complaining of swelling in the left leg which had been present since 1916. Examination

Results of Functional Tests

Case	Age	Sex	Upper Tone Limit		Rinne Test (256)	Schwabach Test (256)	
			Air	Bone			
1	59	M	R 10,000	10,000	—	+ 2	
			L 8,000	9,000	—	+ 4	
2	50	F	R 11,000	.....	—	+13	Pink promontory
			L 10,000	.....	—	+ 7	Pink promontory
3	59	M	R 10,000	12,000	+	— 2	
			L 10,000	12,000	+	Normal	
4	42	M	R 15,500	16,500	+	Normal	Pink promontory
			L 14,500	18,500	+	Normal	on right ride

revealed an anterior bowing of the tibia on the left side and enlargement of the vertex of the head. In 1932 he suffered from subacute iritis. Roentgen examination revealed lesions in the skull, tibia and right humerus. The changes in the skull were described by the roentgenologists as follows: "The entire frontal bone and the anterior half of the left temporal and the inferior half of the right temporal bone are extensively infiltrated by a lesion involving the diploe and apparently the center table of the skull." Biopsy of a specimen from the tibia on March 30, 1931, confirmed the diagnosis of Paget's disease.

The Wassermann reaction of the blood was negative; the calcium and the phosphorus content of the blood were normal.

The patient had experienced tinnitus for about five years but no vertigo. He had a dull headache occasionally. In 1918, while suffering from influenza, he had otitis media on the right side for three months; this was followed by good hearing. There was no familial history of deafness.

Examination of the ears, nose and throat gave essentially negative results except for a mobile atrophic scar in the upper posterior quadrant of the left ear drum.

The results of the examination of hearing are shown in figure 3.

CASE 4.—M. S., aged 42, appeared at the clinic on Dec. 9, 1933, complaining of a gradual change in posture for thirteen years, with bending of the right forearm and tibia and an increase in the size of the head.

Physical examination revealed a marked anteflexion of the spine and bowing of the right forearm and of both legs. Roentgen examination revealed extensive involvement of the skeleton with Paget's disease. This was confirmed by biopsy on December 15. The roentgenologist stated on Nov. 8, 1933: "This is the most spectacular case of Paget's disease ever seen in the clinic. The bones of the skull are enormously thickened, and the normal trabecular pattern is entirely lost, being replaced by a coarse pattern made up of ragged patches of increased density interspersed with areas of amorphous decreased density."

The Wassermann reaction of the blood was negative; the calcium and the phosphorus content of the blood were normal.

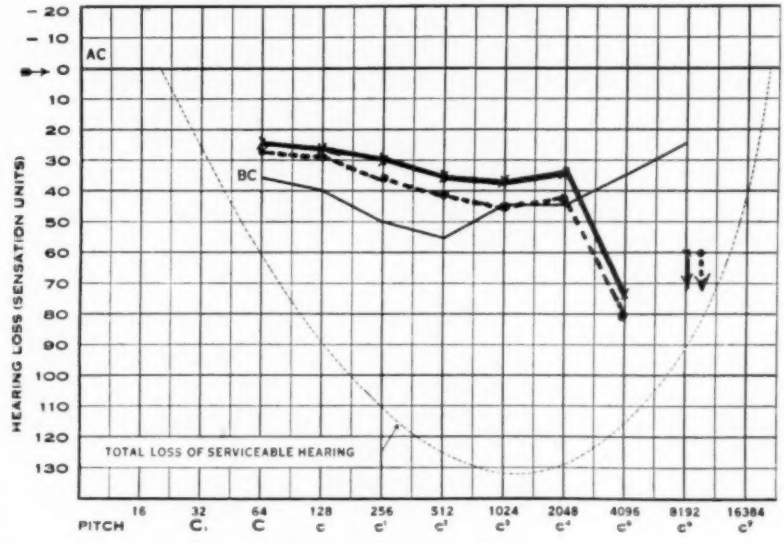

Fig. 1 (case 1).—The solid line represents the threshold of hearing in the right ear; the broken line shows the threshold in the left ear. In this and in the following figures the line *AC* indicates normal hearing and the line *BC* indicates the reading of the audiometer for a person of normal hearing tested with the D = 80,904 bone conduction receiver.

In March 1934 the patient noted a fulness in the right ear, with some impairment of hearing but no tinnitus or vertigo. He had had no previous aural trouble, and there was no familial history of deafness. Examination of the nose and throat gave essentially negative results except for the enlargement of the bones of the face. The left drum and canal appeared normal. The right drum was dull and presented a pinkish hue in the region of the promontory. There was a striking lack of any evidence of acute inflammation in the nose, throat or nasopharynx. The fulness persisted for a month, after which it was found that the pinkish color of the promontory was diminished and that the impairment of hearing had undergone a change. While the fulness was present, the reaction to the Rinne test

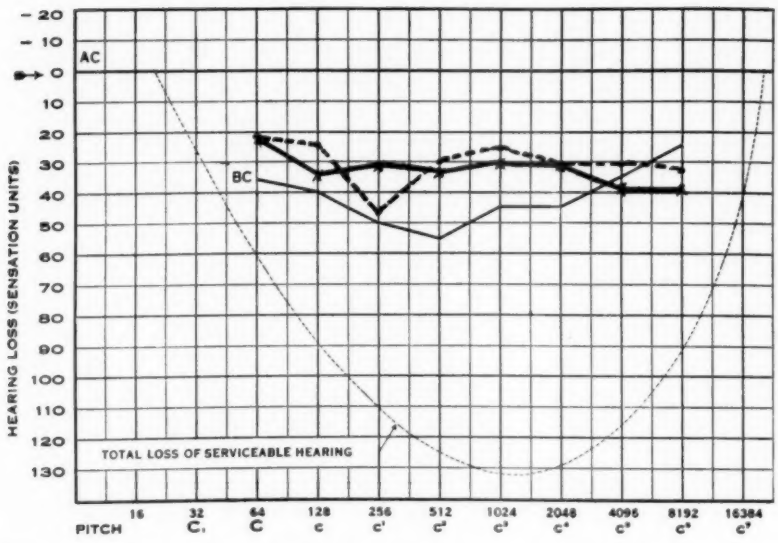

Fig. 2 (case 2).—The solid line represents the loss of hearing in the right ear; the broken line shows the loss of hearing in the left ear.

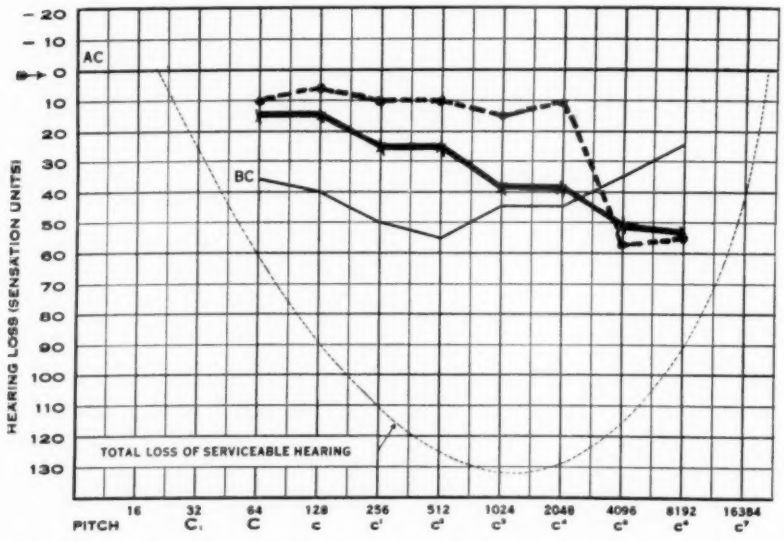

Fig. 3 (case 3).—The solid line represents the loss of hearing in the right ear; the broken line shows the loss of hearing in the left ear.

on that side was negative, and in the Weber test the sound was referred to the same side. The reaction to the Schwabach test was normal in the left ear and +2 in the right ear. The whispered voice test showed adequate response at 15 feet (4.57 meters) on both sides but less for high frequency sounds as represented by spoken words "six" and "seven." By June 15, 1934, when the fulness in the right ear had disappeared, the sound was not referred to that ear in the Weber test, and the response to the Rinne test was positive and that to the Schwabach test was normal. Hearing was tested again on Dec. 28, 1934, and on Nov. 1, 1935. The findings on the latter date are shown in the table. The response of the labyrinths to the caloric test with cold water was within normal limits.

The audiograms for the four patients are shown in figures 1, 2, 3 and 4. The results of functional tests are given in the table.

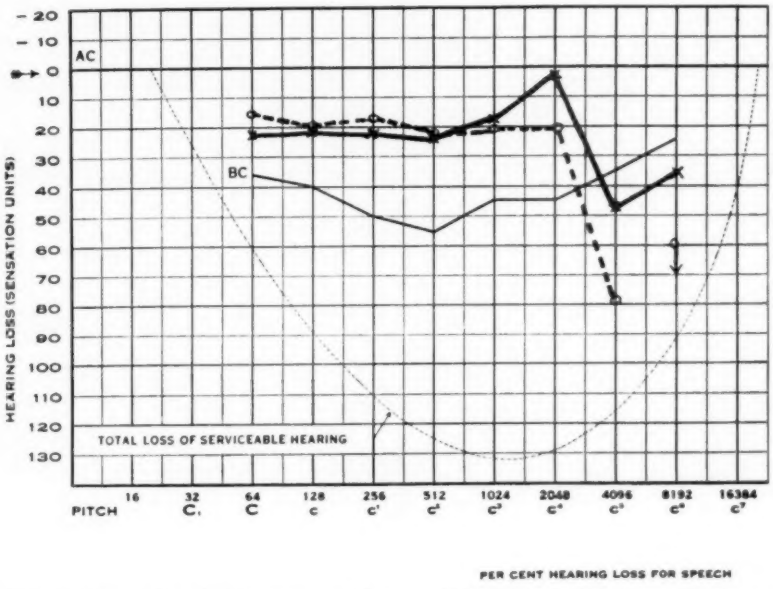

Fig. 4 (case 4).—Audiogram on Nov. 1, 1935. The solid line represents the loss of hearing in the right ear; the broken line shows the loss of hearing in the left ear.

COMMENT

The upper tone limits shown in the table have been determined by the monochord. The intensity produced by this instrument is, of course, limited, but it is greater than that of the 2-A audiometer at the frequency of 8,192 vibrations per second (as illustrated by cases 1 and 4).

In case 1 the impairment of hearing was almost identical in the two ears. Although there was a loss of conduction, indicated by the results of the Rinne and Schwabach tests, the curve for hearing and the lowered upper tone limits showed that the inner ear or nerve deafness predominated.

The pink color of the promontory in case 2 suggested that there was active disease of the bone in that area on both sides, and the con-

duction deafness could be explained by impairment of movement of the foot-plate of the stapes. On the basis of these findings alone the diagnosis of otosclerosis might have been suggested, and it could not be excluded, but the history of the onset and development of the deafness along with the diffuse involvement of the bones of the skull by Paget's disease was strong evidence that the disease in the capsule of the inner ear was a part of the generalized disease of the skull.

Mr. B. (case 3) exhibited essentially the same characteristics as Mr. C. (case 1) except that the hearing impairment was less advanced. The changes in the skull in case 4 were far advanced before the appearance of symptoms in the ear. At the time of onset of impairment of hearing in the right ear the pink color of the promontory was marked; there were, besides, a negative response to the Rinne test and increased bone conduction. Periodic observation since that time showed a gradual change in the condition. With the recession of the pink color the response to the Rinne test again became positive and the principal loss of hearing was in the high tone range. Although the loss of hearing for high tones by air conduction was greater in the left ear, the upper tone limit by bone conduction was lower in the right ear.

The most striking characteristic in the four cases reported were the greater loss of hearing in the high tone range and the lowered upper tone limit. In the first two cases also there was definite loss of conduction while in cases 3 and 4 the well preserved bone conduction suggested some impairment of sound conduction. (In the case of conduction deafness it is probable that the acuity of hearing by bone conduction influences the threshold for low tones by air conduction as determined by the audiometer.)

These findings correspond fairly well with those in the cases summarized by Barth as regards the type of deafness. In none of the cases in our series, however, was there evidence of any symptoms referable to the vestibular apparatus.

The precise manner in which the inner ear or nerve deafness was produced in these cases cannot be determined from the clinical study alone. Clinical observations indicate, however, that Paget's disease of bone does not produce impairment of hearing except in patients with extensive disease in the bones of the skull, including the temporal bone. The diffuse nature of the changes in bone has been demonstrated in cases in which the histopathologic studies have been reported. Some evidence has been presented by Otto Mayer [1] to explain the nerve impairment on the basis of involvement of the branches of the auditory nerve by the formation of new bone.

The findings in cases 2 and 4 of our series present some evidence that the conduction impairment may be brought about by changes in bone in the promontory involving the annular ligament or the foot-plate of the stapes.

It is evident from these cases and from the cases previously reported that osteitis deformans is capable of producing conduction deafness as well as inner ear deafness but that the inner ear or nerve deafness is the most common characteristic.

The possibility that otosclerosis might be present along with osteitis deformans must be recognized. On the basis of functional tests alone differentiation may not be possible, but when the whole clinical picture is considered, certain points are evident which may serve to differentiate between the two conditions.

Although there may be certain points of similarity both in the local histopathologic picture and in the type of hearing impairment between otosclerosis and osteitis deformans involving the temporal bone, a consideration of the history and progress of the two diseases brings out a definite contrast and appears to indicate that any close relationship is unlikely.

Osteitis deformans usually occurs in late adult life, while otosclerosis rarely has its onset after middle age. Osteitis deformans is usually widespread, particularly in the cases in which deafness is present, while otosclerosis, so far as is known, is localized to the capsule of the inner ear.

The familial tendency of otosclerosis is well recognized, whereas such a tendency is not a characteristic of Paget's disease. The incidence of the two diseases offers a marked contrast. The ratio of the number of cases of otosclerosis to that of Paget's disease in this clinic has been approximately 10:1. If the cases of Paget's disease without impairment of hearing are excluded, the ratio becomes 60:1.

SUMMARY

Of twenty-five patients with Paget's disease, seven showed involvement of the skull. Four of these patients had impairment of hearing. The most common characteristic of the deafness in these cases appears to be an impairment of the inner ear. Two of the patients also showed a marked loss of conduction. One patient showed conduction deafness in one ear during one stage of the disease.[8] These findings correspond fairly well with those previously reported in the literature.

The clinical characteristics as a whole offer little evidence of any relationship between osteitis deformans (Paget's disease) and otosclerosis.

8. Since this report was submitted a fifth patient has been observed with extensive Paget's disease of the skull and impairment of hearing. The functional tests indicated a loss of inner ear or perceptive hearing of mild degree in both ears.

Clinical Notes

SPHENO-OCCIPITAL CHORDOMA

Report of a Case

S. E. Gould, M.D., Eloise, Mich.

Recently several excellent reviews have served to direct attention to an interesting group of tumors, the chordomas. A chordoma is a neoplasm of notochordal origin. It arises from the embryonic remains of the chorda dorsalis, the primitive axial skeleton of a vertebrate, which is derived from the entoderm. The notochordal cells become surrounded by the bodies of the vertebrae and soon are confined to the central portions of the intervertebral disks. Such cells may escape encasement by cartilage during development or conceivably as a result of trauma. Theoretically, a chordoma may be produced at any point along the axial skeleton where notochordal cells are found. The chordomas have been classified as (1) cranial, (2) vertebral and (3) sacrococcygeal. They have been reported as occurring in various locations in the following order of frequency: in the sacrococcygeal region, in ninety-one cases; in the spheno-occipital region, in fifty-two cases; in the cervical portion of the spine, in twelve cases, and in other locations, rarely.

Of the fifty-two reported cases of spheno-occipital chordoma, only twelve have been reported in the American literature. Fletcher, Woltman and Adson [1] reviewed the reported cases of sacrococcygeal chordoma; Hass,[2] those of the cranial chordoma, and Owen, Hershey and Gurdjian,[3] those of chordoma of the cervical portion of the spine. It appears that this type of tumor is not as rare or as ill defined clinically as was previously believed. In each of the locations mentioned the tumor presents a fairly constant symptomatology.

Fletcher, Woltman and Adson, reporting ten new cases of sacrococcygeal chordoma, pointed out the predominant occurrence of the tumor among males, the frequent incidence of trauma, the constant presence of pain and the local destruction of bone, as seen on roentgen examination. They stated that the average period of survival in their series was three and one-half years. They advised surgical resection, decompression of the sacrum and the use of high roentgen therapy to relieve pain, retard the growth of the tumor and prolong the life of the patient. One patient was well nine years after the operation. Owen, Hershey

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From the Department of Pathology, Wayne University, College of Medicine, Detroit, and the Dr. William J. Seymour Hospital division of the Eloise Hospital and Infirmary.

1. Fletcher, E. M.; Woltman, H. W., and Adson, A. W.: Sacrococcygeal Chordomas; Clinical and Pathological Study, Arch. Neurol. & Psychiat. **33**:283 (Feb.) 1935.

2. Hass, G. M.: Chordoma of Cranium and Cervical Portion of Spine: Review of Literature with Report of Case, Arch. Neurol. & Psychiat. **32**:300 (Aug.) 1934.

3. Owen, C. I.; Hershey, L. N., and Gurdjian, E. S.: Chordoma Dorsalis of the Cervical Spine, Am. J. Cancer **16**:830 (July) 1932.

and Gurdjian likewise reported the constant presence of pain in cases of chordoma of the cervical portion of the spine and a picture suggesting tuberculosis of the spine in all cases in which roentgen examination was carried out. In their own case a roentgen diagnosis of metastatic new growth was also considered. The correct diagnosis was made at biopsy at the time of operation. Hass, reviewing fifty-six reported cases of chordoma (forty-seven of cranial tumor and nine of the cervical portion of the spine), reported a proportion of three males to two females and a usual time of the occurrence in the third, fourth or fifth decade, with an average age of 36 years. The symptoms in cases of cranial chordoma are due to impairment of function of the cranial nerves or to compression of the brain stem or spinal cord in the region of the foramen magnum. Frequently a history of trauma is cited. In the cases in which there is involvement of the cranial nerves the duration of life is about three years after the onset of symptoms; in cases of chordoma of the cervical portion of the spine the prognosis is poorest, the period of survival being from two to eighteen months, while in cases of nasopharyngeal tumor the prognosis is variable, depending on the involvement of the brain or spinal cord.

Surgical intervention offers hope occasionally, but in most cases it is followed by temporary improvement, recurrence without metastasis in from six to eighteen months being the rule. High voltage roentgen or radium therapy is sometimes palliative.

Hass gave an excellent, concise basis for differential diagnosis. He pointed out that in cases of chordoma with visual defects suggesting a primary tumor of the hypophysis there is no evidence of functional disturbance of the hypophysis. Adson, Kernohan and Woltman [4] suggested the possibility of recognizing a characteristic roentgen picture as an aid in the preoperative diagnosis of chordoma. At present the preoperative diagnosis has been made in practically all cases at biopsy, tissue for which can be obtained in a case of sacrococcygeal or nasopharyngeal tumor.

As a group, the chordomas are slow-growing, of poor cellular differentiation, malignant by position rather than by cell type, locally invasive and destructive, rarely metastasizing and difficult of complete surgical removal, recurrence being the rule, and they do not respond markedly to radium or high voltage roentgen therapy. The roentgen picture is characteristically one of destruction of bone. Microscopically, chordoma must be differentiated from two common types of tumor, myxochondroma and colloid carcinoma of the intestinal tract.[5]

REPORT OF CASE

J. C., a man aged 68, was admitted to the Dr. William J. Seymour Hospital on Aug. 17, 1934, complaining of a growth in the nose, frequent headache and bleeding from the nose.

In February 1932 the patient noted a growth in the left nostril. In November he was admitted to the New York City Cancer Institute Hospital. A roentgenogram of the skull showed no abnormality. The growth did not respond to surgical intervention, and he was given a course of high voltage roentgen therapy. On Feb. 3, 1933, a specimen of the mass in the nose was removed for biopsy, and

4. Adson, A. W.; Kernohan, J. W., and Woltman, H. W.: Cranial and Cervical Chordomas: Clinical and Histologic Study, Arch. Neurol. & Psychiat. **33**:247 (Feb.) 1935.

5. Ewing, J.: Neoplastic Diseases, ed. 3, Philadelphia, W. B. Saunders Company, 1928, p. 212.

30 millicuries of radium was applied for forty-eight hours. The biopsy revealed a benign epithelial polyp. The growth, however, continued to enlarge, and during the summer of 1933 the patient was unable to breathe through his nose and had frequent attacks of nosebleed and severe headaches in the frontal region. He refused to permit an operation. Since then the attacks of nosebleed and headache had become more frequent and severe, and during the past year he had lost 26 pounds (11.8 Kg.).

Eight years previously he fell and sustained a fracture of the skull.

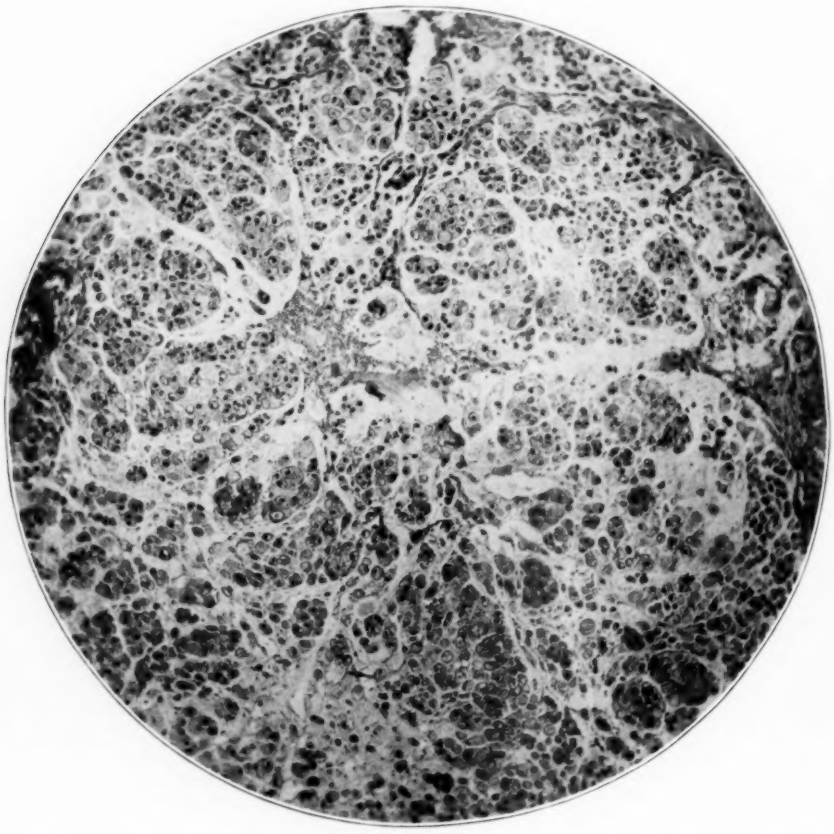

Fig. 1.—Photomicrograph of a spheno-occipital chordoma; magnification, × 60.

Examination revealed the conjunctivae to be injected and the lower lids to be reddened, thickened and slightly everted. There was slight bilateral exophthalmos. The nasal orifices were filled with, and completely obstructed by, soft friable polypi, which bled easily on manipulation. The septum had been eroded by the extensive growth of the polypi.

Roentgenograms showed slight clouding of the frontal sinuses, apparently due to thickening of the lining, and nearly complete opacity of both antrums; of the ethmoid group of sinuses, only a portion of one sphenoid cavity remained clear. There was loss of bone in the lateral wall of the left antrum, probably due to a previous operation.

The Kahn reaction of the blood was negative. On August 22 the blood count showed 3,000,000 red cells and 13,850 white cells, and on September 10, 3,630,000 red cells and 8,950 white cells.

A specimen removed for biopsy on August 20 showed several irregularly shaped masses of mucus-containing tissue which were pink, soft and friable. Microscopic sections stained with hematoxylin and eosin showed large, globular neoplastic cells the cytoplasm of which was pale-staining. The cells had eccentrically placed, well stained round nuclei. Some cells contained two, or even three,

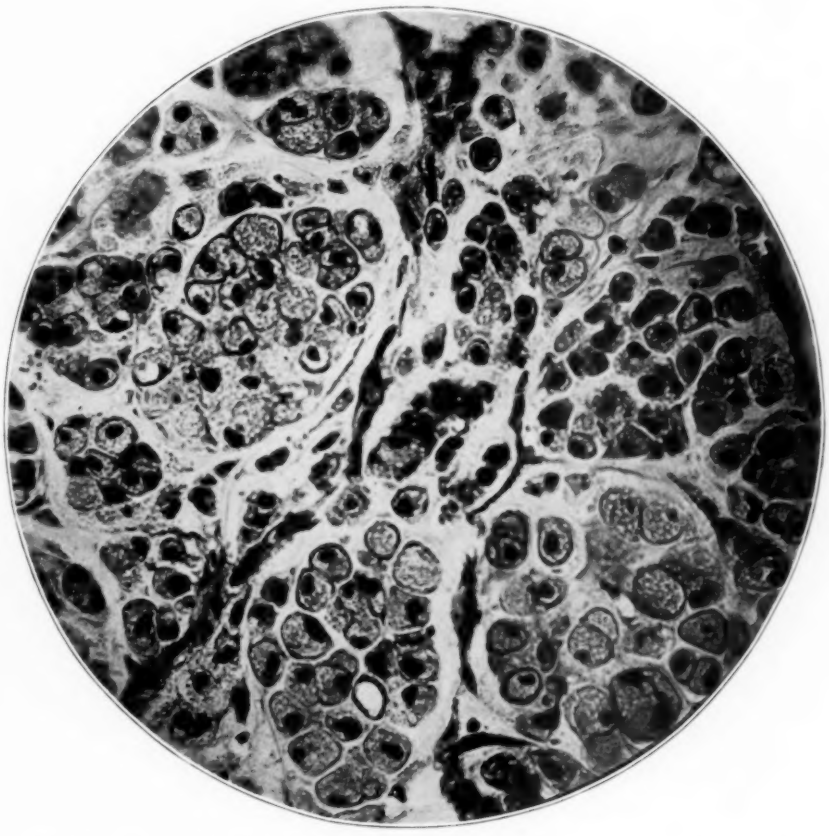

Fig. 2.—Photomicrograph of a spheno-occipital chordoma; magnification, × 258.

nuclei. There was from moderate to abundant intercellular material, which had a mucinous appearance and in some areas resembled fibrous cartilage. The section showed good vascularity and some areas of hemorrhage.

The patient became progressively weaker. Hemorrhage from the nose became more frequent and severe and necessitated repeated transfusions. Surgical treatment was not permitted. From October 25, when a severe chill developed, to his death on October 30 there was a septic temperature, sometimes as high as 104 F.

Autopsy was performed four days after death. There was advanced generalized postmortem autolysis. The brain showed slight edema but no hemorrhage.

Around the optic chiasma in the region of the pituitary gland and the sella turcica there was a yellow-gray exudate covering an area of about 4 cm. in this region of the brain. Around the pituitary gland there was an exudate of thick, yellowish-gray pus, and a probe easily entered the sphenoid sinus. The neighboring bony structures were removed by breaking through the bony plate anterior to the sella turcica, through the wings of the sphenoid bone along the middle cranial fossa and posteriorly through the occipital bone just above the foramen magnum. The soft structures of the nasopharynx were dissected as far forward as they could be reached, and the bony specimen was removed. The upper portion of the nasopharynx was occluded by a friable, jelly-like pink mass, which extended from the sphenoid sinuses well down into the pharynx and eroded the ethmoid cells.

The diagnosis was malignant chordoma of the spheno-occipital region invading the sphenoid and ethmoid sinuses and the nasopharynx.

Microscopic sections of the growth were similar to those observed at biopsy.

SUMMARY

A case of spheno-occipital chordoma is reported. The patient was a man, aged 68, whose first symptom was the appearance of a growth in the nose. Biopsy two months before death established the diagnosis. Surgical intervention, radium and high voltage roentgen therapy did not affect the tumor. Death occurred two years and nine months after the first symptom and was due to purulent basilar meningitis, the result of erosion of the sphenoid with infection from the nasopharynx. The neoplasm extended locally and widely and did not metastasize.

A history of fracture of the skull five and one-half years before the onset of symptoms is etiologically of interest because of the possibility of the escape of remains of notochordal cells at the spheno-occipital articulation and their subsequent proliferation as a result of trauma.

A NEW HEAD BAND

J. GUY JONES, M.D., DALLAS, TEXAS

The presentation of a new technic instrument or appliance or of a modification of a standard type of instrument or appliance should be withheld until the value of the innovation has been proved. After several years of personal use and after the receipt of many letters of praise from others, I feel justified in presenting for consideration a new type of head band.

The bands in general use do not fit the curvature of the head; in front the band rests principally on the lower segment because of the weight of the mirror. This does not favor the firm fixation of the mirror or comfort to the wearer during prolonged use.

Study of the accompanying illustration gives a clear idea of the design of the new head band. The band is cut so that the upper border is shorter than the lower, the segment having been cut from a sphere instead of from a plane surface. The mirror may be fixed before either eye at the wish of the surgeon, but, unless otherwise specified, it is designed to be fixed before the left eye. The latest models have an extra brad placed just above the stud holding the

mirror, thus giving an extra firmness and fixation of the mirror and prolonging the usefulness of the band.

There are two difficulties which I have been unable to overcome to obtain an ideal head band. Like all bands, this one is uncomfortable to wear in warm weather.

A new head band.

Furthermore, the band is not porous or waterproof, so that perspiration from the forehead and oil from the scalp and hair gradually change the color of the leather, and after prolonged use the band becomes rancid.

These bands are priced at $2.75 each and may be obtained from the Tipton Supply Company, 3116 Live Oak Street, Dallas, Texas.

A TRANSILLUMINATOR FOR THE EYES AND THE ACCESSORY NASAL SINUSES

LOUIS H. SCHWARTZ, M.D., NEW YORK

The apparatus consists of a handle, *H*, with a push switch, *C*, which lights both bulbs. Two bars, *B* and *B*, are mounted on the end of the handle at right angles.

A B

Fig. 1.—The transilluminator in use.

Two light carriers fit on the bars in such a way that they can be separated, as shown in figure 1 *A*, or brought together, as shown in figure 1 *B*. The lamps are under the metal caps, *A* and *A*, which are removable. When the transilluminator is used for examination of the maxillary antrums, a glass shield, *G*, is slipped

over the lights. *T* indicates the cord tips for connecting the apparatus to a battery or current controller.

The instrument possesses the following features:

1. It permits simultaneous transillumination of both frontal sinuses or both ocular globes.

2. When one lamp is placed under each pupil the ophthalmologist is able to compare readily the cornea, pupil, iris, lens or anterior chamber of one eye with the other.

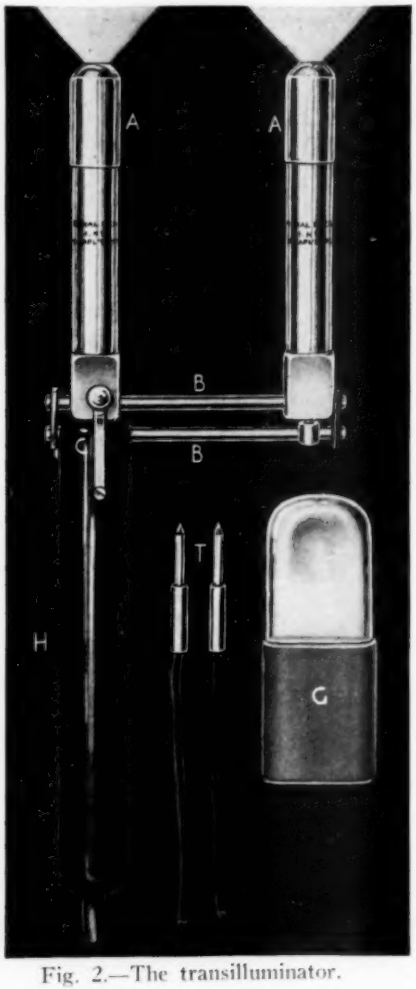

Fig. 2.—The transilluminator.

3. Ordinary flash light bulbs are used.

4. The only parts which require sterilization are the metal caps, *A* and *A*, and the glass shield, *G*.

5. It can be used safely and efficiently in any part of the body where transillumination is advantageous.

6. It is simple and has no complicated, costly parts to get out of order.

Abstracts from Current Literature

Ear

Methods of Testing the Hearing in Infants and Young Children. C. C. Bunch, J. Pediat. **5**: 535 (Oct.) 1934.

In this critical review methods for testing the hearing of small children are discussed. Definite advances have been made in this field during the past ten years, especially in the development of the audiometer. Some knowledge of this subject is particularly valuable to the pediatrician. The history of tests of hearing of school children is briefly reviewed, and the various methods for testing hearing are described and evaluated.

LeMaster, Evanston, Ill. [Am. J. Dis. Child.]

Treatment of Disorders of the Internal Ear. R. Panse, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **139**:244, 1935.

Panse maintains that in cases of disorder of the internal ear the exact localization of the disease process is extremely difficult and that it is generally impossible to determine where between the fenestrae and the cerebrum the disease is located. The therapeutic results likewise are unsatisfactory and are usually limited to a mitigation of such symptoms as subjective sounds and vertigo. The author thinks that an effective method of treatment has been greatly neglected in recent years, namely, the use of the *rarefacteur* of Delstanche. This apparatus was originally operated by hand, but with the attachment of a motor it has become an instrument that influences not only the tympanic membrane, the chain of ossicles and the fenestrae but also the internal ear with its lymph channels and the auditory nerves. The author gives a tabular report of twelve cases in which the treatment proved helpful, and he says that he could cite many more cases. Editor's Abstract.

Function of the Cortical Auditory Centers: Results of Studies by Means of Pavlov's Conditioned Reflexes. M. Eliasson, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **139**:246, 1935.

Eliasson reviews the literature on the function of the cortical centers, showing that considerable advance was made possible by means of Pavlov's method of studying conditioned reflexes. In giving a brief outline of Pavlov's method of research he explains the difference between unconditioned and conditioned reflexes. Whereas unconditioned reflexes are inherited, conditioned reflexes are formed by each individual animal. This capacity of animals to form conditioned reflexes is of great importance for their well-being. Conditioned reflexes may have a secretory or a motor effect. The author points out that Pavlov experimented chiefly with the salivary conditioned reflex. Before describing his own experiments the author explains such terms as ordinary and accessory (extraordinary) stimuli and main and accessory reflexes. Eliasson conducted his experiments on dogs. First he made various types of experiments on normal dogs; then these same dogs were subjected to total or partial extirpation of the temporal lobes. After these interventions the experiments were repeated. When conditioned reflexes had been formed in the animals by means of various sound stimuli, experiments were made with different variations. Tests with sounds of different pitch revealed that there was increased salivation not only in response to ordinary tones but also in response to extraordinary tones; however, there was a quantitative difference in the secretion. It was found that the further removed the extraordinary tone was from the ordinary tone, the less was the secretory response. Prolonged training of the

animals narrowed the conditioned reflex down to a single tone; that is, in the terminology of Pavlov, "differentiation" was achieved. After pointing out that the studies of Bieliakow and Krasnogorskij revealed that this differentiation is due to a process of inhibition, Eliasson continues the description of his own experiments, on the basis of which he reaches the conclusion that the cortical auditory center is apparently the highest inhibition center for the function of hearing. Its partial destruction is followed by changes in all functions that are connected with the processes of inhibition; that is, there is an increase in the conditioned sound reflexes, and their effacement is more difficult. Differentiation, which is based on a process of inhibition is retained, but it loses some of its perfection; for instance, when large portions of the cortical auditory center were destroyed, the differentiation of complex sound stimuli proved impossible, although the efforts were continued for three years. Eliasson points out that the observations of other investigators in Pavlov's laboratory contradict Munk's theory regarding the presence of special convolutions in the auditory region for the perception of sounds of a certain pitch. It was found that in case of the preservation of small remnants of the cortical auditory center the capacity for the analysis of sounds of different pitches remains unimpaired. A differentiation of certain regions of the auditory center for the analysis of complex sound stimuli is considered possible by Pavlov. In this connection the author mentions experiments in which after partial extirpation of the auditory center a tone had lost its action as a constituent of a chord but had retained it as an independent stimulus.

On the basis of numerous experiments, Pavlov reached the conclusion that elements of the sound analyzer are distributed in the cortex and even in the hemispheres; they have the capacity only of elementary synthesis and analysis of the sound stimuli. Pavlov is of the opinion that the auditory analyzer is distributed among the other analyzers "far beyond the region hitherto assumed."

EDITOR'S ABSTRACT.

PERCEPTION AND CONCEPTION, PARTICULARLY IN REGARD TO HEARING. O. GOEBEL, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **139**:264, 1935.

Goebel shows that external stimuli produce changes in the nerves which, to a certain degree, resemble the stimuli. For instance, in the fibers of the optic nerve something like light flows; in the fibers of the acoustic nerve something sounds. The author emphasizes that the "something" is similar to, but not identical with, the original stimulus. Light and sound do not stimulate the optic and auditory nerves directly; it is necessary for the external stimulus to produce in the sense organs a process which makes oscillations and their conduction possible. The author shows how this is done in the auditory cells as well as in the rods and cones of the retina. The conduction of light and sound oscillations outside the nerve fibers is impossible because the stimulation of especially constructed molecules must be involved. If these molecules are absent there is no possibility for corresponding atomic or electric oscillations. If in the sensory nerves, particularly in the optic and auditory nerves, external impressions are conducted in the manner of the corresponding molecular or electrical stimulus, light and sound stimuli must be elicited also after extirpation of the cerebrum. In this connection the author points out that a hen that has been deprived of its brain becomes startled by noises and avoids near obstacles. Decerebrate monkeys likewise react to sounds. In human subjects similar observations have been made, for in case of cortical blindness, resulting from a tumor of the falx cerebri in the region of the occipital lobe, the light reflex of the pupils is intact. The author emphasizes that only conscious perception is dependent on the cerebrum. To be sure, in human subjects the cerebrum dominates the body to a much greater extent than is the case in animals.

The question is raised whether the different ganglion cells of a perception center perform only one form of intramolecular movement or whether many forms may develop in them. There is evidence that each ganglion cell of the cerebral hemispheres works with a definite intramolecular movement. In view of

the enormous number of different auditory nerve fibers, it seems inconceivable that the ganglion cells and fiber networks of the auditory center should be capable of executing this unlimited number of intramolecular movements.

The author shows further that the "perception centers" are connected with "inhibition centers." Whenever the tonus of these inhibition centers is especially high, perception ceases. To concentrate attention means to increase the tonus of the inhibition centers of the perception centers that are not needed in the observation. It may be assumed that the inhibition centers have a certain permanent tonus, and if it is weakened or abolished, perception becomes increased, for instance, hyperacusis develops.

The stimuli taken up by the perception centers pass directly to the pertaining "memory spheres," so far as these are capable of perception, that is, uninfluenced by the inhibition centers. The memory centers retain the external stimuli. The author further discusses two possibilities of the structure of the "main remembrance center" for auditory impressions. Then he shows that for the development of a conception not only the main remembrance center for auditory impressions is necessary but also the remembrance centers for other types of perceptions (optic, olfactory, etc.). He shows that the development of a conception is an automatic process. In the various memory centers processes develop that are similar to the real perceptions that were active in the first development. In many instances numerous repetitions of the same processes are necessary until a certain perception or recollection reproduces the pertaining memory images.

In the last part of his paper the author gives especial attention to the memory center for auditory impressions. He shows that it consists of two distinct spheres, the chief sphere retaining noises and the secondary sphere retaining musical sounds. All sound impressions are communicated to both parts, but the memory center for noise retains the noises and all sound impressions that are not pure, particularly speech, including, of course, its pure tones. The center for sound memory retains music and songs.

EDITOR'S ABSTRACT.

OTIC GANGLION. P. J. MINK, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **139**:283, 1935.

Mink describes an observation on himself which directed his attention to the otic ganglion and led him to study the anatomy and physiology of this structure. He found that the recent textbooks of otology either mention the otic ganglion briefly or disregard it entirely, and he thinks that the neglect of this ganglion is partly responsible for the chaos that is encountered in the physiology of hearing. The ganglion was first described by Arnold in 1831, and Mink largely follows Arnold's original description and even reproduces an illustration that was first made by Arnold. After giving a description of the anatomy and function of the otic ganglion he states as his conclusion that the otic ganglion is an autonomous motor center for the organ of hearing.

EDITOR'S ABSTRACT.

ACUTE MASTOIDITIS AND NEPHRITIS. K. LÖWY, Monatschr. f. Ohrenh. **69**:690 (June) 1935.

Löwy analyzes 2,047 cases of acute otitis in order to determine the relationship between diseases of the ear and nephritis. He summarizes his conclusions as follows: 1. In a patient with renal disease the postoperative course of otitis is usually normal. 2. In approximately 0.7 per cent of cases of acute mastoiditis there is hemorrhagic nephritis. 3. In some of these cases otitis is the introductory infection; in some it is the primary focus, and in some it is both. 4. Otogenic nephritis occurs most frequently in children, and many of them have glandular swellings. 5. The postoperative prognosis in cases of otogenic nephritis is favorable so far as the renal disorder is concerned. 6. The existence of hemorrhagic nephritis may under certain conditions be the indication for opening of the mastoid process.

EDITOR'S ABSTRACT.

VISIBLE EFFECT OF FARADIC CURRENT ON THE HEARING OF CHILDREN WHO ARE HARD OF HEARING. E. FRÖSCHELS, Monatschr. f. Ohrenh. **69**:735 (June) 1935.

Fröschels calls attenton to a peculiar behavior observed in many so-called deaf-mutes, namely, that nystagmus-like movements of the eyeballs develop if tones from the pipes of Urbantschitsch are led to the patient by means of a tube. He thinks that this symptom may be regarded as due to vestibular stimulation elicited by acoustic influences. He was able to observe this movement in about 50 per cent of the deaf-mutes. Since he has been in the habit of giving electrical treatments to patients who have severe hardness of hearing, he decided to use the faradic current also in the treatment of children who seemed to be really deaf, in that the aforementioned ocular movements were not elicitable. He gave from fifteen to twenty faradic treatments, each one lasting for from five to ten minutes, and found that in a considerable number of the patients the test with the Urbantschitsch harmonica then elicited the ocular movements or the related swaying movements. Other patients reacted after several series of treatments with the faradic current (separated by intervals of from four to eight weeks). The author cites several cases and then states that the purpose of his report is to call attention to the fact that under the influence of faradization signs may develop that are known to be indications of hearing. He does not discuss the degree of improvement in hearing.

EDITOR'S ABSTRACT.

RADICAL OPERATION THROUGH THE AUDITORY CANAL. F. S. BOCKSTEIN, Monatschr. f. Ohrenh. **69**:776 (July) 1935.

Bockstein shows that a review of the results of radical trepanation of the temporal bone for chronic otitis disclosed that this intervention does not fulfil the expectations. According to some authors the results are satisfactory in only about 20 per cent of cases. However, the author thinks that, in spite of its disadvantages, radical trepanation of the temporal bone is still a great achievement, provided its use is limited to suitable cases. He shows that many of the leading otologists no longer consider it as absolutely necessary for such conditions as cholesteatoma or even for facial paralysis and fistular symptoms in cases of chronic otitis. It is now almost generally accepted that in cases of chronic otitis the operation should be as conservative as possible. After mentioning several otologists who have suggested conservative methods, the author calls attention to the radical operation through the auditory canal, which was described by Hoffmann in 1892. He mentions other otologists who have improved and advocated this method and then reports his own experiences with it in 120 cases of chronic otitis. He uses local anesthesia. In the first group of 50 cases he operated according to the purely intra-aural method, whereas in the later cases he added the external incision of Heermann. He describes both procedures and then discusses the operation on the bone, showing that if the bony auditory meatus is sufficiently wide and straight the opening of the attic or antrum can be effected at once. If this is not the case the auditory meatus must be widened. After this has been done, two ways are possible: Either the surgeon begins with the attic and then attacks the antrum, or he begins with the antrum and enters the attic after the bridge has been removed. The author employed the first procedure, which is essentially Stacke's method, in 72 cases; in the other 48 cases he employed the second method. He describes the conditions for which each method is advantageous. The after-treatment is essentially the same with the intra-aural method as with the ordinary radical operation. The author evaluates the immediate and the late results of the intra-aural operation. For the evaluation of late results only 89 patients (of a total of 120) were available. In 48 (54 per cent) the results were excellent, in that there was complete smooth epidermization and no secretion. In 26 cases the results were good, for there was complete epidermization in the antrum and the attic, but in the tympanic cavity the mucous membrane was preserved and there was either no secretion or only slight mucous secretion without odor; that is, there

was benign mesotympanitis in place of the former epitympanitis. In 9 cases (10.1 per cent) the author designates the results as satisfactory. In the region of the antrum there were incompletely epidermized islands, with periodic secretion and occasionally with accumulation of thickened epidermis, but the cavity was wide open and thus permitted the observation of the process. In 6 cases (6.7 per cent) the results were unsatisfactory in that fetid secretion and granulations were noted. The author concludes that on the whole the results are as good as can be expected after the surgical treatment of chronic otitis.

EDITOR'S ABSTRACT.

THERAPY AND PROGNOSIS IN FRACTURES OF THE CRANIAL BASE WITH INVOLVEMENT OF THE EAR. H. LOEBELL, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folia otolaryng.) **26**:117 (June) 1935.

After outlining the symptomatology of fractures of the cranial base, Loebell points out that involvement of the ear can be demonstrated by a discharge of blood or cerebrospinal fluid from the ear and by examination of the hearing capacity and of the apparatus of equilibrium. Further he stresses the importance of roentgenoscopy of the cranium according to the method of Stenvers and Schüller or occasionally of E. G. Mayer or of Altschul-Uffenorde and also bitemporal roentgenography. In doubtful cases these methods will help in deciding whether the ear is involved and in clarifying the problem of the complicated fracture. However, the author admits that a roentgenogram which does not disclose a fracture does not definitely exclude a fracture. After citing the stastistics of other observers on the incidence of involvement of the temporal bone he discusses the observations he made in eighty-five cases of fracture of the cranial base. He thinks that in uncomplicated cases, whether the ear is closed or open, an expectant attitude is advisable. In borderline cases of fracture with nonfebrile and painless suppurations (whether the suppurations existed before the accident or developed later) and in which there is practically no inflammation, it is advisable to take an observant and conservative stand. If a suppurating ear is involved in the fracture, if complications, including secondary suppurations, threaten or are present, the complete opening of the spaces of the middle ear with wide exposure of the dura and of possible foci of hemorrhages is the method of choice. When these rules were followed, the author had no fatality from fractures of the cranial base, and he was able to save even patients with severe meningitis who otherwise would probably have died. In patients with severe injuries, deafness and disturbances in equilibrium may persist, but in the majority these subside gradually. A definite estimation of the amount of permanent impairment is not possible before at least one year has passed. The author thinks that the correctness of his method of treatment of fractures of the cranial base is attested by cases of severest nasopharyngeal hemorrhages, of spontaneous tamponade of the sigmoid sinus, of fracture of the cranial base in the presence of a suppurating cavity, of late meningitis and of the resumption of flying by two gliders who had sustained fractures of the cranial base.

EDITOR'S ABSTRACT.

Larynx

NEW TREATMENT FOR PARALYSIS OF THE LABIA VOCALIA AND FOR RESPIRATORY DISTURBANCES IN BILATERAL PARALYSIS OF THE RECURRENT NERVE. E. FRÖSCHELS, Monatschr. f. Ohrenh. **69**:641 (June) 1935.

Fröschels points out that an operation for goiter is occasionally followed by paralysis of the recurrent nerve. This is not necessarily caused by surgical injury of the nerve but may be the result of disturbances in the blood supply. If there is bilateral disturbance of the nerve, there is frequently fixation of the labia vocalia in the median or paramedian position. The author reports a case in which the left labium vocale was fixed in the paramedian position and the right one in

the cadaveric position. The patient consulted the author on account of complete aphonia four months after an operation for goiter. Since Gutzmann's method of producing a subtonal humming sound elicited no movement of the labia vocalia, the author decided to resort to a method that he had used for the treatment of paralysis of the soft palate. Knowledge of the fact that the power of one group of muscles is transmitted to another group induced him to try pushing movements. For this treatment the hands are placed lightly on the chest and are pushed downward while vowels are uttered. Intonation should not be made too late or too early, but the sounds should not be uttered until the arms are in the downward movement. Under the influence of this treatment the patient's condition improved considerably, and after a year she was able to sing. Laryngoscopy revealed that the paramedian position of the left labium vocale was not changed but that the right labium moved freely in both directions. Before discussing the treatment for the respiratory disturbances in a case of postoperative bilateral paralysis of the recurrent nerve the author describes the earlier treatment, which was never promising. After evaluating the suggestions of several other authors, he reports the histories of four patients on whom he employed the aforementioned pushing exercises with good success. Because of the simplicity of the method and because its results are permanent, he feels justified in reporting this treatment, in spite of the fact that the number of cases is still limited. Whether pushing exercises will be effective in cases of recurrent paralysis of other origins, the author is unable to say.

EDITOR'S ABSTRACT.

Nose

QUANTITATIVE DETERMINATION OF MINERALS IN INFLAMMATORY DISEASES OF THE NOSE AND OF THE NASAL SINUSES. R. MITTERMAIER, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **139**:219, 1935.

Mittermaier points out that former studies with physicochemical and chemical methods demonstrated that inflammatory diseases of the nose and of the accessory sinuses are accompanied with considerable disturbances in the acid-base equilibrium, in the fluid content and in the mineral content. After citing other investigators who have studied these problems, the author points out that in former reports he has stressed the necessity of further studies on the various electrolytes, particularly sodium, potassium, calcium, chlorine and the phosphate radical. In this report he describes quantitative determinations of electrolytes. In all, 800 tests were made on material from 150 patients. The results of the tests are given in tables. One table gives the mineral contents of the blood serum, another shows the mineral contents of the tonsils and of the nasal mucous membrane and still others show the mineral contents of pus and mucus from a number of patients. One graph records the chlorine content of the secretion in the course of temporary improvement; another gives the chlorine and sodium values during temporary improvement of a pathologic process of the maxillary sinus. The mineral contents were watched also in the course of the treatment of patients with acute or subacute inflammation of the accessory sinuses. The author concludes that if the information with regard to mineral metabolism of patients with inflammatory disorders is taken as a basis, the results obtained in these studies largely corroborate the expectations, for instance, the increase in potassium in suppuration. On the other hand, these tests demonstrate also that it would not have been correct simply to apply the knowledge of mineral metabolism in general to this special field.

EDITOR'S ABSTRACT.

SURGICAL THERAPY IN SEVERE MANIFESTATIONS OF CHRONIC SINUSITIS. T. DAITO, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **139**:300, 1935.

In the cases described by Daito the severe symptoms of chronic sinusitis were not the result of isolated disease of the maxillary sinus but rather of an involvement of several sinuses (pansinusitis). In some cases the disorder was localized in

groups of cells not detectable by inspection or by exploratory irrigation. Roentgenoscopy, with due attention to all details, is therefore absolutely necessary. In case of retention of pus (pyocele) the symptoms are most severe. However, even in case of edema or of catarrhal changes of the sinusal mucous membrane the symptoms may be severe. Opening the maxillary sinus often does not counteract the symptoms, such as a feeling of heaviness in the head, girdle sensation around the head, a feeling of fulness or pressure on the nasal dorsum, impossibility of mental concentration and so on. Surgical cleaning out of the sinuses, however, generally causes these symptoms to disappear completely. The incision at the orbit should, if possible, be only as short as for probatory opening of the frontal sinus, according to Killian, and should not go beyond the eyebrows. The author emphasizes that this intervention is not followed by atrophy and that, if possible, the median concha should be preserved. Roentgenograms revealed that the large postoperative cavity later becomes filled with cicatricial tissue. The mucous membrane of the sinuses was found more or less diseased, even in cases in which this condition had only been suspected and had not been corroborated by inspection or irrigation. This indicates that severe frontal sinusitis and ethmoiditis may exist and may be the source of rhinogenic symptoms without corresponding rhinoscopic changes. Occasionally, the inflammation manifests itself only by an edematous swelling. In case of retention of pus the symptoms become more severe. The author recommends the more frequent use of the orbital route for a radical operation on the frontal sinus, for with this approach even cavities that extend far laterally can be thoroughly cleared out and a favorable cosmetic result can be obtained.

EDITOR'S ABSTRACT.

THERAPY OF OZENA WITH VIOSTEROL AND ERGOTAMINE TARTRATE. L. RIEGELE, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folia oto-laryng.) **26**:146, 1935.

Riegele followed Glasscheib's suggestion that because of a deficiency of cholesterol in the blood of patients with ozena, viosterol should be administered. However, although the viosterol had a favorable effect on ozena, it did not produce a complete cure, and it was decided that the medication with viosterol had to be supplemented with some other substance. Since it had been observed that patients with ozena frequently exhibited symptoms referable to the autonomic nervous system, such as increased sweating, tachycardia and acrocyanosis, ergotamine tartrate was used, for it is known to exert a favorable effect on sympathicotonic symptoms. The author employed the combination treatment of ozena in the following manner: Every morning and evening the patient was given 1 dragée (0.6 Gm.) of viosterol, and after the noon meal, 1 tablet (0.001 Gm.) of ergotamine tartrate dissolved in a glass of warm water. Local treatment, in the form of nasal irrigations, was entirely discontinued. The patients were subjected to a control examination every two weeks. Observations on over thirty patients who were subjected to this treatment revealed that treatment with viosterol and ergotamine tartrate produces encouraging results. In the period between the first and the fourth month, increased secretion from the mucous membrane gradually detaches the scabs. In the beginning the mucous secretion is still fetid, but later the mucus becomes thinner and the fetor disappears. About the time when the crusts become detached, the blood perfusion of the mucous membrane has usually considerably increased. The mucous membrane becomes thicker. If this swelling reaches the region of the median concha, severe frontal headaches may develop, which can usually be controlled by suitable remedies. This period lasts about two weeks. During the last two (fifth and sixth) months of the treatment, the secretion becomes gradually lessened. After six months the medication is discontinued, but in case of recurrence the treatment is resumed for a short time. The general condition of the patients is often considerably improved, the appetite increases and fatigue disappears. This favorable effect on the general condition the author considers as an especial advantage of this treatment.

EDITOR'S ABSTRACT.

NONSPECIFIC THERAPY OF VASOMOTOR RHINITIS. L. RIEGELE, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folia oto-laryng.) **26**:148, 1935.

Riegele describes the clinical histories of several patients with vasomotor rhinitis in whom he obtained favorable results without intranasal intervention. His treatment consists in producing an intracutaneous wheal by means of a mixture of cresol which is free from protein. The wheals are made at two day intervals until from six to eight have been made. In trying the treatment on himself the author found that about eight hours after the first intracutaneous injection the watery secretion and the intranasal swelling disappeared, but on the following day slight swelling again developed. However, after six intracutaneous injections the nasal breathing remained free. After describing several other cases in which similar results were obtained, the author concludes that, although vasomotor rhinitis is apparently limited to the nose, it yields to general nonspecific shock therapy, that is, to measures that reduce the pathologically increased tonus of the sympathetic nervous system. The nonspecific shock therapy was effective in some cases in which other measures had failed. The author considers that this injection treatment produces a nonspecific desensitization. EDITOR'S ABSTRACT.

HEMATOMA AND ABSCESS OF THE NASAL SEPTUM AND THEIR COMPLICATIONS. W. COMBECHER, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folia oto-laryng.) **26**:156, 1935.

Combecher thinks that it is entirely justifiable to consider hematoma and abscess of the nasal septum together, since a sharp differentiation between the two is often difficult. An abscess frequently results when a hematoma becomes complicated by a secondary infection. The cause of a hematoma or of an abscess is usually traumatic. The lesion develops as the result of the impact of a force from the outside (thrust, blow or fall) or after an endonasal intervention. Hematoma may result after any operation on the nasal septum, but it is most frequent after a submucous resection. The author describes the mechanism of the development of traumatic hematoma and traumatic abscess and calls attention to a third cause of abscess of the nasal septum, namely, inflammation. Such an abscess may originate in the nose, in the external skin or in the teeth. The author explains these different modes of pathogenesis, giving special attention to the dental origin, which he illustrates with two case histories. Then he shows that, as regards the symptomatology, a hematoma and an abscess of the nasal septum show considerable similarity. As a rule, the outside of the nose is reddish and swollen. On closer inspection, a spherical tumor is visible in both nasal openings. This fluctuating tumor is attached to the septum by a broad base and usually obliterates the nasal passage so that nasal breathing is impossible and the patient cannot close the mouth for any length of time. The pharynx dries out, the patient snores, the speaking voice is dull and nasal, and headaches, fever and chills develop. These symptoms permit the diagnosis of hematoma or abscess, but the differentiation of the two conditions necessitates an exploratory puncture. After describing the usual clinical course of an abscess of the nasal septum the author gives a statistical review of twenty-three patients who were observed at his clinic between 1926 and 1934. Two cases are described in detail, because meningitis developed, but both patients recovered. In discussing the pathogenesis of meningitic complications the author considers the blood-borne infection, transmission by way of the lymph channels and the possibility of a mechanical spreading of the pus to the lamina cribrosa. He shows, further, that an abscess of the nasal septum may result also in osteomyelitis. In discussing the prevention of these complications the author states that conservative methods of treatment are inadequate in the treatment of hematoma of the septum. As soon as a hematoma or an abscess has been detected, opening and drainage are necessary. After the drain has been removed prolonged tamponade is usually advisable in order to prevent nasal deformity.

EDITOR'S ABSTRACT.

Miscellaneous

METHODS OF DEMONSTRATION AND CLINICAL ASPECTS OF THE MITOGENETIC RADIATION OF BLOOD. W. HERMES, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **139**:236, 1935.

Hermes, after reviewing the literature on mitogenetic radiation and giving especial attention to the characteristics of the mitogenetic radiation of the blood, points out that Heinemann applied Gurwitch's original observations clinically. He took from different patients extravasated blood that had been stained like lacquer by the addition of magnesium sulfate and used it as the sender. Yeast that had been grown in beer wort was used as the detector. With the aid of disks that are moved by hand, he interrupted the radiation and thus produced a better effect. Since this apparatus of Heinemann was difficult to operate, Grahe and Weidemann constructed a device in which the disks were moved by means of a motor. Hermes used this device for his studies and reaches the conclusion that the Grahe-Weidemann apparatus is useful in the determination of the mitogenetic radiation of blood. In order to find the optimal exposure time and the optimal number of interruptions of radiation, the author tried various arrangements and found that the induction effect is best at five minutes and that forty interruptions per minute are most favorable. Tabular reports of the results of tests on different persons revealed that the blood of a considerable number produced no radiating effect. In this connection the author points out that Heinemann observed the absence of the mitogenetic radiation particularly in the blood of patients with carcinoma, cachexia, chronic tonsillitis, generalized infections and so on and that Fischer observed the same in the blood of syphilitic patients. The author, however, observed the absence of mitogenetic radiation also in some young and entirely normal persons and consequently warns against interpreting the absence of mitogenetic radiation as a sign of disease.

EDITOR'S ABSTRACT.

Society Transactions

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T. C. Galloway, M.D., *President*

Walter H. Theobald, *Secretary*

Primary Adenocystic Carcinoma or Cylindroma of the Trachea: Report of a Case. Dr. M. R. Guttman.

A basal cell cancer of rare type (adenocystic carcinoma or cylindroma) is reported as a primary growth in the trachea, bringing the total number of recorded primary malignant neoplasms of this organ to 28.

Histologically such a neoplasm exhibits anastomosing cords and nests of slightly anaplastic cells that closely resemble basal cells of surface epithelium. The cell nests present central lumens that may contain cellular débris or a hyalin-like material.

Grossly the neoplasm tends to grow slowly and to infiltrate beneath the tracheal mucosa. The lumen of the trachea is irregularly reduced eccentrically. The mucosa overlying the growth appears red and congested. There is no tendency for the neoplasm to erode the mucosa or to metastasize to regional lymph glands or to the abdominal viscera.

The characteristic symptomatology concerns a progressive increase in dyspnea associated with otherwise apparent good health.

The characteristic roentgen finding is an irregular constriction of the tracheal lumen by a submucous infiltrate.

Bronchoscopy reveals a smooth irregular constriction of the tracheal lumen due to a submucous infiltrate that feels firm to the exploring tip of the bronchoscope. The portion of the mucosa overlying the infiltration is red and congested, while the noninvolved portion may be normal in appearance.

The diagnosis can be established definitely only by biopsy.

The treatment consists in cautious administration of high voltage roentgen radiation with due regard to the tracheal cartilage. For the dyspnea tracheotomy is indicated.

The prognosis as to ultimate cure is doubtful, although the life expectancy can be reckoned in years.

DISCUSSION

Dr. Ernest A. Bredlau: I did not have an opportunity to read Dr. Guttman's excellent paper until a few moments ago, and so I will confine myself to a brief discussion of the general class of tumors called cylindroma, among which the tumor of Dr. Guttman's patient was placed. I think the tumor which Dr. Guttman has described should be classed as a basal cell carcinoma, adenocystic in type. I had an opportunity to study a similar tumor which originated in the nose, arising from the septum. I do not think that the tumor I observed or the one which Dr. Guttman has presented represents the cylindroma. The cylindroma has epithelial nests which are surrounded by a definite hyaline sheath. Since Billroth described this kind of tumor and called it cylindroma, tumors that have small hyaline spaces or spaces filled with granular débris and which have epithelial tissue around them have been classified as cylindroma. The origin of such tumors is still in question. A few authors still believe them to be endotheliomatous, but most workers, I think, now consider them as epithelial. Krumpecker clarified

the subject and called such tumors basal cell carcinomas. He divided basal cell carcinomas into three types, adenomatous, cystic and solid. He succeeded in tracing them to the basal cell layer of the skin and mucous membrane. The cylindroma of Billroth, he places as a subvariety of the basal cell carcinoma. Clinically the basal cell carcinomas all present similar pictures. They are all mildly malignant, have a tendency to recur and seldom penetrate the surrounding tissue. For an inaccessible growth such as that in Dr. Guttman's case the only treatment is that with radiation, but if the tumor is accessible surgical removal followed by irradiation is, I believe, better, as quicker results can be obtained thus.

Dr. Joseph C. Beck: I cannot let this opportunity go by without bringing out an important clinical point. Histologic discussion is splendid, but the clinical picture that interests me is that this woman had the courage to travel by airplane from Oklahoma to Chicago with the condition which Dr. Guttman described—and she was in desperate need of air when she arrived. It was interesting to hear her say that she felt better while in the plane, so one need not be reluctant about sending patients, particularly those suffering with foreign bodies and other disorders, by air.

Dr. Bredlau pointed out that some growths of this type should be treated surgically. In the present case the tumor involved the cartilage, and surgical removal would have caused complete collapse of the region involved, so my associates and I felt that it was better to use radiation therapy.

Theories of Hearing and Recent Experimental Work. Dr. Arno B. Luckhardt and Dr. R. W. Gerard (by invitation).

This article is not for publication. No discussion followed the reading of it.

An Evaluation of Tuning Forks for Use in Routine Practice and Some Comparisons with Audiometers. Dr. Robert Sonnenschein.

The most important tests made with tuning forks are described and the types of audiometers in common use are listed. The subject matter may be summarized as follows:

Objections Advanced Against the Use of Tuning Forks.—1. Practically all tuning forks when struck give forth not only the fundamental tone but a number of overtones. With piano strings the first overtone is an octave higher than the fundamental tone, but with tuning forks the first overtone has a frequency of more than six times that of the fundamental tone. However, when one is using the higher-pitched forks these overtones do not interfere since the first overtone of c-4 (2,048 double vibrations) and that of c-5 (4,096 double vibrations) are higher than most persons can hear, and therefore patients are not disturbed by them.

2. With the lowest-pitched or even medium-pitched forks weights may be applied, but these shorten the period of vibration. However, if the fork is properly calibrated, shifting of the weights will give a range of from five to seven tones for one fork, thus in many instances eliminating the need for several forks in the lower ranges. Instead of heavy weights, a rubber band placed on each prong near the distal end will eliminate the overtones and at the same time not shorten the period of vibration.

3. There is a lack of uniform intensity due to difference in excitation. This is really no serious objection, because each fork has its own rate of decrement, and experiment shows that unless there is a very great difference in the force employed in striking the fork there is no essential difference in its rate of vibration.

4. One cannot get the same curve showing the loss of hearing figured in sensation units as with the audiometer. This is not a valid objection, since the "constant" of decrement may be had for each fork, and then, using the formula suggested by Harvey Fletcher, one may obtain the same curve as with the audiometer, but the work will require a somewhat longer time.

Advantages of Tuning Forks Over the Audiometer.—1. The tuning forks are easy to manipulate, especially the light weight magnesium-aluminum alloy forks produced by Mr. B. E. Eisenhouer at the Riverbank Laboratories.

2. There is no need of adjustments of the appliances.

3. The forks will serve indefinitely unless grossly abused or mishandled.

4. A sufficient number, especially of the light weight forks, may easily be carried about to be used at the bedside or otherwise away from the office or clinic.

5. The expense is comparatively low; the cost even of the complete series of forks is much less than that of the standard audiometer, and the cost is really insignificant when only the four or five forks essential for all tests are purchased.

Information to Be Derived by Testing with Tuning Forks.—1. The range of hearing by air from c-2 (16 double vibrations) to c-5 (4,096 double vibrations) may be determined.

2. The duration of bone conduction as compared with a normal standard may be demonstrated by means of the Schwabach test, with the fork on the vertex, or by using the bone conduction factor of the Rinne test, with the fork on the mastoid process.

3. The relation of air to bone conduction in the same patient may be found.

4. Lateralization of sound, if any, may be determined by means of the Weber test.

5. Malingering of unilateral deafness may be unmasked.

6. A definite localization of the lesion is possible; that is to say, the observer may discover whether the involvement is mainly one of the conduction apparatus or one of the perception apparatus or a combination of the two. This is the most important contribution of the tuning fork tests.

Advantages of the Audiometer as an Instrument of Precision.—1. Graphs may be obtained showing the loss of hearing figured in sensation units, and the figures can be converted into percentage of loss of hearing.

2. The records obtained may be compared with those made by others or with other records in a series of tests over certain lapses of time, so that progress or changes in the state of hearing may be noted.

3. The data obtained are very valuable for medicolegal work, especially in cases in which there is a question of industrial compensation.

4. Audiometers, especially the Western Electric 1-A, are eminently suited to the purposes of research.

5. The Western Electric 42-A Test Set is said to be suitable for the detection of malingering.

Objections to the Use of the Audiometer.—1. Great expense is involved. Thus far the standard types have cost in the neighborhood of $600 each, and the Western Electric audiometer 1-A, I believe, has been priced in the neighborhood of $1,500 or $2,000. However, recently some very low-priced audiometers have been placed on the market, and if they prove entirely satisfactory this factor will be greatly lessened.

2. Audiometers lack easy portability, as they are extremely heavy.

3. Unless "beat" tuning is employed, great care must be observed in watching the battery, etc., so that the findings may be uniform and accurate.

4. There is still some difference in the calibration of the various types of audiometers. To make records properly comparable, a standard unit should be employed.

5. The curves obtained do not always indicate the actual type of lesion present.

6. Unless the new types are thoroughly tested in acoustic laboratories to determine their accuracy with reference to intensity, etc., uniformity in reactions will not be possible.

7. It has been held by some authors that the audiometer has one distinct drawback, namely, that in testing children up to 6 and 7 years of age with it one cannot get accurate responses.

Summary.—In the testing of the function of hearing all available aids should be employed. It is necessary that a careful clinical history be elicited not only with reference to the state of hearing but with regard to any systemic pathologic condition or condition localized in another part of the body. The patient should be observed with reference to his attitude, efforts at lip reading, loudness of voice and other factors, and then a complete examination of the ears, nose, nasopharynx and pharynx carried out. Although the voice cannot be standardized, if unaccentuated whisper or conversation is employed with proper precautions a practical estimate of the hearing is obtained. This, together with all the data previously mentioned and the results from the use of tuning forks, especially for the Weber, Schwabach and Rinne tests, enables one to make a diagnosis relative to the location of the lesion affecting hearing, if any.

With various types of audiometers graphs may be obtained showing the loss of hearing figured in sensation units. These may likewise be obtained with tuning forks when one is supplied with a constant of decrement. For the highest tones neither the forks nor the audiometers suffice, and the Edelman-Galton whistle or, better still, the monochord is needed.

In a word, when properly employed, both forks and audiometers give most important information. Each type of appliance has advantages and some disadvantages. Merely having an audiogram without the clinical history and a complete examination of the patient is of little value, and it seems to me, therefore, that audiometery should be carried out only by otologists in their clinics or private practice, not by laymen in the effort to prescribe hearing aids.

While audiometers are desirable for use in the testing of the function of hearing and for research they are, in my opinion, not absolutely essential so far as diagnosis is concerned, for the diagnosis may be established with proper use of a few good forks in making the more important tests.

DISCUSSION

Dr. George E. Shambaugh Jr.: The advantages and limitations of the audiometer have been well brought out by Dr. Sonnenschein. The chief advantage of the audiometer is that it supplies a convenient standard yardstick by which the hearing for a series of tones can be measured and charted and the chart fairly accurately compared with subsequent ones obtained at later dates. One advantage which Dr. Sonnenschein did not mention is that the audiogram is not subject to the psychologic variations accompanying the whisper test; particularly when the whisperer is a physician overoptimistic about his own treatments for deafness.

The most serious limitation of the usual audiogram is that it does not indicate the location of the lesion. To be sure, a sharp down curve of the hearing for the higher tones is often seen in cases of nerve deafness while a horizontal type of curve is usually seen in cases of conduction deafness, but there are so many exceptions, as I shall show, that the type of curve cannot be relied on for diagnosis. Another limitation is that the usual audiogram represents a combination of air and bone conduction, so that a unilateral dead ear will not be detected by a study of the audiogram, while a false picture will be obtained concerning the hearing for low tones in patients with conduction deafness.

The value of the audiometer for making clinical tests of hearing has recently acquired new interest because the aggressive commercial concerns marketing hearing devices have begun to picture themselves as "aural optometrists" and are doing their own audiographic work on prospective customers. These salesmen would like to call themselves audiometrists, persons who test the hearing and prescribe an appropriate device just as optometrists examine the vision and prescribe glasses. I can see many serious objections to this practice and no advantages. Experience has shown that persons with conduction deafness are

usually helped by an electrical device because, even with the usual earpiece, there is considerable bone conduction imparted to the head. Persons with nerve deafness, on the other hand, are very rarely benefited by an electrical device. In other words, the decision as to whether or not an electrical device should be advised depends mostly on the type of deafness present, and this cannot be diagnosed accurately from the audiogram. Some day there may be hearing aids available which amplify certain tones more than others, and then an audiogram may be of value to indicate which tones are most deficient and which type of device will serve. But at the present time there is only one type of instrument put out by each concern, and the instrument can be adjusted only in the amount of amplification which it offers. The prescribing of electrical aids to hearing at the present time, therefore, depends chiefly on the diagnosis of conduction deafness, and this cannot be diagnosed by the usual audiogram.

A much more serious objection to the development of a profession of audiometry is that patients who need and can be helped by medical attention will inevitably be missed, whereas if a competent otologist makes the tests the true condition will be detected and cured.

Let me illustrate these various points by some cases seen in the past year or two (lantern slides):

Slide 1: This audiogram represents a patient who has normal hearing in the left ear and a moderate defect in the right ear due to beginning ankylosis of the stapes from otosclerosis.

Slide 2: This one is that of a patient who has primary nerve deafness in the right ear and a dead ear on the left as revealed by the tuning fork tests. The apparent hearing in the left ear is by bone conduction to the opposite ear. The audiogram is not dependable in the detection of unilateral severe deafness, because of this element of bone conduction to the other ear.

Slide 3: The patient has bilateral conduction deafness. Notice how the hearing by air conduction is gone for the tone of 32 double vibrations, and yet the standard audiogram shows hearing for this tone, because of the element of bone conduction.

Slide 4: The patient has nerve deafness due to congenital syphilis.

Slide 5: The patient has nerve deafness. By comparing bone conduction with pure air conduction it is possible to diagnose the type of deafness with the audiometer alone. However, these tests are very arduous and take a long time. The same information is gained in a few moments with the usual tuning fork tests.

Slide 6: This is the audiogram of a man aged 37. Nerve deafness, unlikely to be helped by an electrical aid, one would say from the audiogram. The tuning fork tests, however, show no hearing for the 32 and 64 double vibration forks, the Rinne test is strongly negative and the Schwabach test prolonged. In other words, this patient has conduction deafness. He uses an electrical device with benefit.

Slide 7: This is the audiogram of a man aged 39. The curve suggests conduction deafness, which he has. He should be helped by an electrical device.

Slide 8: Here is an audiogram which is almost identical with the one just shown, and yet the patient has nerve deafness and will not find an electrical device of much value.

The audiogram curve cannot be relied on to indicate the type of deafness.

Slide 9: This is the audiogram of a patient, aged 49, who had been deaf for twenty years. He has a completely deaf ear on the left side with an attic cholesteatoma which produced severe attacks of dizziness several years ago. The audiogram shows apparent hearing in this ear owing to bone conduction to the right ear. The audiometrist would miss the cholesteatoma, which has already destroyed the labyrinth and which, if untreated, might cost this patient his life. The audiometrist would probably diagnose conduction deafness in the right ear from the horizontal type of curve, and he would be correct in this instance. But, examination disclosed a mass of adenoid blocking the right eustachian tube, and after this was removed, at the age of 49, this patient's hearing returned to normal and has remained normal ever since.

Slide 10: The young man whose audiogram is shown here, the son of a prominent lawyer, had been hard of hearing for two months, unable to hear a whisper from farther than 3 feet (91 cm.) with one ear. The audiogram is consistent with conduction deafness, but the tuning fork tests revealed nerve deafness. A routine Wassermann test, which should be taken in all cases of primary nerve deafness, was positive. It then became apparent that a stubborn rectal ulcer occurring months before, which took many weeks to heal, had been a chancre. Antisyphilitic treatment resulted in normal hearing two months later. Syphilis would never have been suspected by the audiometrist and would have remained undiagnosed until aortitis, paresis or tabes developed years later.

Slide 11: This audiogram represents a woman aged 50, deaf for several years. The tuning fork tests revealed nerve deafness. She also showed a pasty, pudgy complexion; her hair and eyebrows were thin; her reactions were very slow; her feet and hands were puffy and always cold, and she was sleepy all day long. A metabolic test (which, of course, would never be ordered by an audiometrist) showed a basal rate of —24. Under thyroid medication the hearing returned to normal, and the symptoms of myxedema disappeared.

Slide 12: The patient, a man aged 54, had been somewhat hard of hearing for twenty-five years—worse since influenza two months before the test. The audiogram suggests conduction deafness, but the audiometrist would not have seen that both tympanic cavities were filled with clear yellow serum. After this had been removed by paracentesis and inflation, the hearing improved to its previous level. There is still moderate nerve deafness but not enough to interfere seriously with his carrying on his usual business.

Slide 13: A young woman aged 26 had been increasingly deaf for five years. The audiogram certainly looks as though she had otosclerosis, and the audiometrist would have prescribed a bone conduction hearing device, price $125. This patient is a maid who receives a salary of $8 a week. Examination disclosed a mass of adenoids blocking the left eustachian tube. Removal of the adenoids resulted in normal hearing in this ear at a fraction of the cost of the bone conduction device.

Slide 14: A woman aged 38 had been deaf since the occurrence of scarlet fever at the age of 9. The right drum membrane was thickened; the left was absent. The placing of a Toynbee artificial ear drum in the left ear resulted in a remarkable improvement in hearing. This patient would have been helped by an electrical device but how much better off she is with the artificial ear drum!

Slide 15: A man aged 56 had been deaf for three months following several attacks of vertigo, nausea and vomiting. He was seen during his third attack of vertigo. The audiogram suggests conduction deafness, but the tuning fork tests showed inner ear deafness and diplacusis, the forks being heard several tones higher in the affected ear. A diagnosis of exudative labyrinthitis probably due to a focus of infection was made and an infected pair of tonsils removed. The hearing began to improve within a week and is now normal with no diplacusis and no further attacks of vertigo. This diagnosis could not have been made from the audiogram alone.

Slide 16: A music teacher aged 59 had been deaf for two months with diplacusis, which prevented her from listening to music, and with loud roaring tinnitus. The audiogram suggests conduction deafness, but the tuning forks revealed nerve deafness with diplacusis, the forks being heard from several notes to an octave higher in the affected ear. A diagnosis of exudative labyrinthitis was made, and search for a focus of infection revealed suppurative ethmoiditis, which had been present for about a year. This cleared up after about a dozen treatments by Proetz displacement, and soon the hearing returned to normal, the roaring became almost imperceptible and the diplacusis disappeared. Here again the diagnosis could be made only on the basis of the tuning fork tests.

In conclusion I may say that the audiometer is a convenient means of obtaining records of the hearing acuity for a series of tones to compare with future records. The usual audiogram cannot be used to diagnose the location of the lesion; it gives a false picture of the hearing for low tones in persons with conduction deaf-

ness, and it will not reveal a dead ear. The prescribing of a hearing device depends chiefly on the diagnosis of the type of deafness, and this can be arrived at only by the tuning fork tests. The audiometer, at the present time, is of little practical value in prescribing hearing devices and the use of it, unless accompanied by a complete otologic examination, will inevitably result in the neglect of many types of deafness that could be cured.

DR. SHERMAN L. SHAPIRO: The society is to be congratulated on taking up this subject, which has needed ventilation for some time. I enjoyed listening to the reading of the paper of Dr. Sonnenschein and the discussion by Dr. Shambaugh. I especially wish to commend Dr. Shambaugh for illustrating so clearly the limitations of the audiometer. It is, to my mind, regrettable that enthusiasts have filled the literature with contributions on the virtue of this instrument, producing the impression that it constitutes the last word in scientific apparatus for the testing of hearing. The fact is that the results obtained by the use of the audiometer relate to a mixture of air and bone conduction and, whatever their value from a research or other standpoint, they certainly have no relationship to fundamentals of otology as taught hitherto. If the accurate and unfailing diagnosis of the type and true extent of the hearing defects is to be the criterion by which this instrument is to be judged I can consider it to be only a pseudoscientific apparatus.

I think it behooves this society to put itself on record definitely as to what the real value of the audiometer is and who should use it. I might add one more comment: Emphasis has been placed on the value of the audiometer in estimating the percentage of disability in medicolegal cases. The percentage of loss as estimated in sensation units by means of the audiometer is no more related to the ability to carry on a conversation, which is the practical criterion of deafness in medicolegal cases, than is a similar computation made from the results of tests with tuning forks, neither figure bearing a constant ratio to the patient's capacity for hearing the voice; the audiometer would be even less true in this respect than tuning forks because of the impossibility of testing pure air conduction with it. Furthermore, in Illinois at least, partial losses of hearing are not compensable if the suit is against an employer, compensation being awarded only for total conversational deafness.

DR. SAMUEL SALINGER: I should like to have said everything Dr. Sonnenschein and Dr. Shambaugh said and probably would do so had I their knowledge, experience and forensic ability. I heartily endorse what they stated regarding the use of the audiometer by laymen. I think it is comparable to allowing lay persons to set themselves up as advisers in matters of health because they possess a laboratory or an x-ray apparatus. If this presentation has done nothing else, it has brought to the foreground the necessity of combating the promiscuous use of this device.

DR. ARNO B. LUCKHARDT: I have had no experience with the audiometer except as I used it in some research, reports of which I have not published, but I was greatly interested in both of these papers. The great advantage of the audiometer was stated to be that one would have an exact record of the patient's hearing based on tones, which would be valuable in treating the patient and in determining from time to time whether the hearing had improved or not; and if the patient went to another part of the world and consulted another physician he could take with him the audiometric records, and the physician in the new locality would know what his previous difficulty had been and the extent of it on the basis of the records. I am convinced by Dr. Sonnenschein that one can establish a quantitative record by the use of the tuning forks, but is it ever done? I was not quite convinced by him that one gets in successive tuning fork tests the same intensity of sound, for one might strike the tuning fork harder at one time than another. I think there might easily be a different intensity at different tests. If, however, they are constant from time to time, and if one can put down data on the record that are fairly quantitative, I see no necessity for having such an expensive apparatus as the audiometer.

DR. R. W. GERARD: I have never used an audiometer but am reminded of the reply that is often made when any one says "Figures cannot lie"; namely, "Figures do not lie, but liars figure." I wonder how much of the feeling evidenced tonight is against the audiometer, and how much against the audiometrists. The speakers have pointed out many things about the audiometer which I am glad to learn. I am not sure that it can be regarded as a "pseudoscientific" instrument; more evidently, pseudoscientists try to use it without any of the training that otologists bring to bear. I feel that its advantages are those that have been brought out, and I agree that no one but an otologist should attempt to diagnose aural conditions, with or without an audiometer.

DR. AUSTIN A. HAYDEN: I wish to express my appreciation of the program that has been presented this evening. I was particularly interested in the presentations made by Dr. Sonnenschein and Dr. Shambaugh. I was interested in the other papers but not very familiar with their subjects.

The truth about audiometers and tuning forks must lie somewhere in between the very definite statements that have been made in favor of one and against the other. I cannot agree with what has been said against such careful observations as those of Edmond Fowler of New York, who has done outstanding work in the conservation of hearing and the estimation of hearing. His work is accredited almost everywhere in the United States except Chicago. Whether Chicago is right or not remains to be seen, but as I look back over these various discussions I can remember a time when the gentleman whose efforts I consider the "last word" in tuning fork technic, Dr. Robert Sonnenschein, was much more opposed to audiometric examinations than he is now, judging from his paper this evening —and I think his paper was excellent. It contained a very fair summation of the advantages and disadvantages of both methods of testing.

In regard to the statement that quantitative tests can be made by the correct use of tuning forks, that such tests can be made has long been known and no one attempts to deny it. On the other hand, Dr. Sonnenschein has taught at Rush Medical College for many years, and I dare say one can count on the fingers of the hand the number of men from that college who make quantitative records of hearing with tuning forks that can be classed with those obtained with the 2-A audiometer, to say nothing of the 1-A audiometer. I assume in talking of audiometrists—and I regret their advent as much as any one here—that they are just as unqualified to use tuning forks as they are to use audiometers. It is no more an infringement of the law of any state to test hearing by tuning forks than by audiometers, but I think in only one instance have I known an audiometrist, or hearing aid salesman, to make any extensive use, or any use at all, of tuning forks; so it seems to me the use of the apparatus, whether it is pseudoscientific or not, is open to some question. The use of audiometers will operate in favor of making more hearing tests, and whether these tests are as accurate as any that can be made with tuning forks or not they will at least be more frequent.

I was interested in what Dr. Luckhardt said about the permanent uniform records of hearing loss. I think there has been great need for such records. Whether or not a curve is true, at least it is uniform, and even more can be said about that obtained with the 4-A audiometer, which registers the two voices, the female and male, by phonograph. I think it is impossible for a speaker to speak twice in the same tone, at the same pitch or with the same intensity of voice. That must be taken into consideration. Another thing, the physical characteristic of tuning forks is that their maximum loudness is apparent immediately on their being struck; with the audiometer that statement can be reversed. If there is any question as to whether a patient hears or not, the test can be repeated up and down the scale, so that a very accurate record can be made.

I believe, as Dr. Sonnenschein has well said and as Dr. Shambaugh has well echoed, that in the examination of ears and of hearing acuity the entire patient must be taken into consideration, that the very best facilities that medicine has to offer must be used that every instrument whether it is a tuning fork, a mono-

chord or an audiometer has a place in this examination, and that it is greatly to be deplored that any other than fully qualified observers are attempting to enter the field. I believe that otologists who do not use the audiometer seriously handicap themselves. In the past few years there has been built up a demand for such observation. It should always be supplemented by complete tuning fork tests and a physical examination. In the end the one who has "built the best mouse trap" should survive. Whether that will be the audiometrist—and I cannot see how that can be—or the otologist—and I can well see how that can be—I cannot say, but I can say that the subject of hearing was up to the last ten years sadly neglected.

Dr. Howard C. Ballenger: I fully agree with Dr. Sonnenschein's objections to the use of the audiometer and believe that Dr. Shambaugh's experience coincides. However, the quantitative measuring of hearing that my associates and I at Northwestern University have been doing, which involves a great amount of work, would not be possible without the audiometer simply because of lack of energy and of time. It would not have been possible for us to carry on without it, so I believe that the audiometer has a very definite and valuable function in the measurement of hearing. In diagnosis the use of the instrument is supplementary to the clinical examination and the use of the forks.

Dr. Alfred Lewy: One of the points not brought out in favor of the audiometer is that in detecting malingering when a certain amount of hearing remains the best way to trap the malingerer is by making repeated audiometric tests. I think no one is smart enough to respond in exactly the same way to repeated tests.

A good point brought up by Dr. Hayden was in regard to the great propaganda for the sale of audiometers. This has resulted in awakening the public to the problems of deafened persons; in that respect it has been of great benefit.

Along the line of Dr. Shambaugh's presentation I wish to say that it is my opinion, based on a number of experiments, that the loss of hearing represented by the audiometer is not an accurate index to the loss of hearing for speech. I have recently had two high school boys who were smart enough to give good audiograms, but both boys have so great a loss of hearing that they are candidates for special schools for the deaf; 50 per cent loss was all that was indicated in their audiometric records. I also believe that the audiometer is entirely useless for determining a small vestige of hearing. For this purpose the speaking tube is better and often discloses areas of hearing that may be useful in teaching speech.

Dr. G. Henry Mundt: I am one of those who stand in the middle ground. The expense of the audiometer has been mentioned. The buying of a set of Edelman forks was a great expense to me. There is no question in my mind but that a man should use both the audiometer and tuning forks. Dr. Shambaugh is practicing otology as it should be practiced, with both the tuning forks and the audiometer. As Dr. Sonnenschein said, it is possible to build a curve with tuning forks, but it is very tedious and hard to do. In a physical examination one does not argue as to whether one should estimate the glycosuria or study the blood sugar—one does both. And why is it not equally right to use both the audiometer and tuning forks? In my experience the audiometer is a very satisfactory instrument for work with children, certainly as satisfactory as tuning forks, although it is said that it is of little value with children. That may be true, but neither are tuning forks the acme of perfection in making an estimation of hearing defects.

I certainly agree that there is being built up a group of audiometrists. I think that is a very nice term to use, but I think they should be discouraged. I suspect that if one analyzes the reason for the development of such a group, as was suggested by one of the other speakers, one will find that the practice of medicine is responsible, as the practice of medicine is responsible for nearly all of its troubles. I make a plea to you not to say that the audiometer has no value or that tuning forks have no value—one makes both a blood count and a microscopic examination, and I see no reason why one should not use both tuning forks and an audiometer.

DR. W. E. GROVE: I do not wish to enter into polemics about tuning forks and the audiometer, because I use both, but the point brought out about the audiometer—that the record is uniform and can be compared with records from some other part of the country—is erroneous. Nothing can be further from the truth. The audiometer must be standardized from time to time. One's own audiometer will not remain standard for any length of time, and as for giving records that may be compared with records from another portion of the country, the audiometer has no particular advantage.

DR. ROBERT SONNENSCHEIN: I wish to repeat the quotation I used some years ago when speaking with reference to a certain type of new instrument, namely, "Prove all things, hold fast to that which is good." One should use every source of information that will yield any procedure valuable to the patient. Dr. Shambaugh beautifully demonstrated the fact which I tried to bring out, namely, that in many instances the audiometric curve does not show the pathologic condition which is present. Why there is this great disparity at times, I do not know, except that with the lowest tones it is almost impossible to obtain a sufficient intensity with the audiometer to get an accurate result.

The fact that when the audiometer was first introduced I personally was somewhat skeptical about its value from a practical standpoint has no bearing at this time. I have always felt that everyone is entitled to his opinion as long as he works no harm to any one else. I am always ready and happy to get all possible information, and prepared to change my opinion whenever enlightening facts are brought to my attention. Some of the opinions which I brought out originally were substantiated, while in others I may have been mistaken. My principal objections were: First, audiometers as then constituted were too expensive; second, they were heavy and could not be transported easily; third, they had not at that time any means for determining bone conduction, and that is the key to the diagnosis.

Unless work is done in a soundproof room, every tone is masked by from 20 to 30 decibels because of the noise which is present. However, since the testing of patients is in most instances done in a noisy environment, the conditions being the same, the results are comparable and from a practical standpoint sufficient.

As Dr. Levy has said, it is impossible to tell what the efficiency for the hearing of the voice is by means of the audiometer, and from the practical standpoint the hearing efficiency of an ear depends on its ability to hear and understand the spoken voice. It is necessary to hear within a certain range, and if a person can hear the very lowest and some of the very highest tones, that is, if he cannot hear from 200 and 300 to 6,000 and 7,000 double vibrations, the hearing for voice will be poor.

If a person loses only 10 or 20 decibels of hearing, that makes no practical difference. If there is a loss of from 20 to 30 decibels, it means that one must use a rather loud voice, and if there is a loss of from 50 to 60 decibels, a tremendous volume of voice would be required to make the patient hear. The practicability of the thing is what counts—what the examiner wants to know and what the patient wants to know is whether the latter's hearing is or is not so far impaired that the impairment will interfere with his pursuing his usual work or vocation. When there is a lesion of the nerve present, the electrical hearing aids are usually of no assistance, but with a lesion of the middle ear they are usually of great help.

As to Dr. Mundt's statement regarding the Edelman forks, let me say that they were very heavy and very expensive, but that the new magnesium-aluminum alloy forks are very light and less expensive, and one needs only a few forks for the most important tests.

From a practical standpoint, let me say that it is not absolutely necessary to make an audiometric examination, for with a few good forks one can make the diagnosis, know the prognosis and be able to decide on whatever treatment may be available. Audiometers are good adjuncts in the testing of the function of hearing.

Book Reviews

For Stutterers. By Smiley Blanton, M.D., Assistant Professor of Clinical Psychiatry, Cornell University Medical College, and Margaret Gray Blanton. Price, $2. Pp. 186 and index. New York: D. Appleton-Century Company, Inc., 1936.

After a discussion of the basis of speech; the different levels of the mind; the growth of conscience; anxiety; emotional patterns, and the family and the social pattern, there follow a description of stuttering, some current theories, the authors' views on the cause and treatment, other theories of treatment, the prognosis, and a consideration as to how parents, teachers, the public and the stutterer can help overcome this defect.

With others, the authors believe in the emotional etiology of stuttering. The original blocking of the rhythm of speech (stuttering) occurs from mild states of fear, embarrassment or anxiety, reenforced by superimposed conditioned fears. There is need of study in each case of the cause or causes of the anxiety or fear. Stutterers usually suffer from chronic anxiety—"misplaced, misdirected, excessive anxiety about something in the unconscious life which attaches itself to conscious material." The authors do not fully explain the nature of the supposedly underlying emotional difficulties but hint at the freudian conception of unconscious fixation at so-called pregenital or later sexual levels, although they do not use these terms. They believe in the existence of an unconscious mind.

Aside from the establishment of sound physical health and good living conditions, the treatment recommended consists mainly of two methods of psychologic readjustment: (1) freudian psychoanalysis, for a limited number of selected patients, with daily visits of an hour for at least one and, occasionally, two or more years, and (2) individual guidance, which is suitable for the largest number of patients, consisting of the practical application of mental hygiene for from a few weeks to a few months, the frequency of visits depending on the severity.

The prognosis is best in the very young (those from 2 to 4 years old) who just begin to stutter, provided the parents fully cooperate; it is perhaps next most favorable in those from 16 to 22 years old and is worst in those from 10 to 16 years old.

There is much good general information and advice, although some of the views expressed are matters of controversy among the best students of stuttering.

There is hardly any reference to other writers, and no bibliography is given.

Les voies aeriennes et la tuberculose. By G. Portman and H. Retrouvey. Paper. Price, 50 francs. Pp. 312, with 30 illustrations. Paris: Masson & Cie, 1936.

The authors feel that the artificial division of the respiratory tract into two parts, namely the respiratory passages and the lungs, is unnatural. They believe the apparatus should be considered as a whole because of the anatomic, physiologic and clinical interdependence of its various parts and therefore, in this work it is their purpose to effect a collaboration in the interest of the internist as well as the laryngologist.

Although disclaiming any intention of writing a complete treatise on tuberculosis of the various organs comprising this group they nevertheless go into considerable detail describing the clinical forms and pathogenesis of tuberculosis of the nose, sinuses, nasopharynx, pharynx and larynx.

In discussing pathogenesis the authors feel that the various portals of entry have each their own peculiar significance which is difficult in each case to evaluate exactly. However, in the light of experimental as well as clinical evidence they

are becoming more and more convinced that the circulatory systems and particularly the lymphatic apparatus play a predominant rôle.

The chapter on therapy has to do more with the general treatment of tuberculosis than with special treatment of the different organs. All of the accepted measures, such as climate, rest, pneumothorax, biologicals, radiotherapy, as well as the controversial chemical treatment, are described and properly appraised.

The rôle of diseases and obstructions of the nasopharyngeal spaces as predisposing factors to tuberculosis is taken up in a special chapter and stressed in the light of certain statistics bearing on the subject.

Differential diagnosis comes in for considerable discussion, and nontuberculous conditions producing cough, expectoration, loss of weight, etc., such as chronic sinusitis, benign and malignant tumors, foreign bodies and the like are adequately described.

The book is well written and loses nothing through having its subject matter divided and subdivided into numerous chapters and numerical headings. If anything this method of presentation makes for easier reading. All in all it is a creditable piece of work and should be particularly well received by the laryngologist whose knowledge of tuberculosis as a whole has perhaps suffered through too close application to the problem of tuberculosis of the larynx alone.

News and Comment

Scientific Exhibit, American Academy of Ophthalmology and Otolaryngology.—A scientific exhibit will be one of the features of the convention of the American Academy of Ophthalmology and Otolaryngology, to be held in New York on September 26 to October 3. Otolaryngologists who desire space in the exhibit may communicate with Dr. S. J. Kopetzky, 51 West Seventy-Third Street, New York; ophthalmologists, with Dr. Ralph I. Lloyd, 14 Eighth Avenue, Brooklyn.

Directory of Otolaryngologic Societies *

FOREIGN

Collegium Oto-Rhino-Laryngologicum Amicitiæ Sacrum

President: Prof. Gunnar Holmgren, Karolinska Mediko Kirurgiska Institut, Stockholm, Sweden.
Secretary: Prof. Dr. C. E. Benjamins, Verlengde Heereweg, 143 Groningen, The Netherlands.

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Ralph A. Fenton, 1020 S. W. Taylor St., Portland, Ore.
Secretary: Dr. Gordon B. New, Mayo Clinic, Rochester, Minn.
Place: Kansas City, Mo. Time: May 11-15, 1936.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Wells P. Eagleton, 15 Lombardy St., Newark, N. J.
President-Elect: Dr. Frank E. Burch, 408 Peter St., St. Paul.
Executive Secretary: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: New York. Time: Sept. 26-Oct. 3, 1936.

American Bronchoscopic Society

President: Dr. H. M. Taylor, 111 W. Adams St, Jacksonville, Florida.
Secretary: Dr. Lyman Richards, 319 Longwood Ave., Boston.
Place: Detroit. Time: May 27, 1936.

American Laryngological Association

President: Dr. Burt R. Shurly, 62 W. Adams Ave., Detroit.
Secretary: Dr. James A. Babbitt, 1912 Spruce St., Philadelphia.
Place: Detroit. Time: May 25-27, 1936.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Thomas E. Carmody, Metropolitan Bldg., Denver.
Secretary: Dr. R. L. Loughran, Sharon, Conn.
Place: Brown Palace Hotel, Denver. Time: May 18-20, 1936.

SECTIONS:

Eastern.—Chairman: Dr. Henry B. Orton, 24 Commerce St., Newark, N. J.
Southern.—Chairman: Dr. Robin Harris, Lamar Bldg., Jackson, Miss.
Middle.—Chairman: Dr. W. E. Grove, 324 E. Wisconsin Ave., Milwaukee, Wis.
Mid-Western.—Chairman: Dr. Harry W. Lyman, Carleton Bldg., St. Louis.
Western.—Chairman: Dr. Carroll Smith, Paulsen Bldg., Spokane, Wash.

American Otological Society

President: Dr. Francis R. Packard, 304 S. 19th St., Philadelphia.
Secretary: Dr. Thomas J. Harris, 104 E. 40th St., New York.
Place: Detroit. Time: May 28 and 29, 1936.

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* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.